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THE ARTIFICIAL KIDNEY IN THE TREATMENT OF SEVERE PUERPERAL INFECTION DUE TO CLOSTRIDIUM WELCHII, WITH REPORT OF A CASE.

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IN 1947 Kolff published in detail particulars regarding the making and working of an artificial kidney for the treatment of acute renal insufficiency, with the specific intention of enabling others to copy his work. Extracorporeal dialysis had previously been tried by Abel, Rowntree and Turner in 1913. It would appear that, owing to the relatively high cost, the difficulty of production and the infrequency of its use, the artificial kidney has not met with sufficient favour.

The need for an artificial kidney was felt in this hospital when patients suffering from lower nephron nephrosis following trauma had died during conservative treatment for want of a means of tiding them over a temporary period of anuria.

It is the purpose of this article to describe the machine built in Brisbane, and details of the treatment of one patient who almost certainly would have died but for treatment by the artificial kidney. This patient is of interest because she is the first patient reported to have been treated successfully by the artificial kidney in Australia. She is the only woman diagnosed as having a *Clostridium welchii* infection of the uterus in the Brisbane Women's Hospital during the past five years.

Report of a Case.

Mrs. B., aged thirty-six years, was referred to the Brisbane Women's Hospital for supervision of her fifth pregnancy and confinement because of her previous unusual maternal history. Her blood group was O, Rh-negative, and her first two children were normal. However, during the third pregnancy she was found to have anti-Rh antibodies present in high titre, and was advised to undergo premature induction of labour with possible exchange transfusion of the baby. She was not seen again, however, until she was in labour for the third time; the baby died soon after birth with signs of *erythroblastosis fœtalis*. The fourth pregnancy ended in a miscarriage. After the first and second confinements she had had a retained placenta, which was removed manually. At the third confinement the placenta was removed manually as soon as the baby was born.

She reported to the ante-natal clinic for her fifth pregnancy on November 27, 1953, December 4 and December 11, and was found to have anti-Rh albumin-agglutinating antibodies in a titre of 1:256 and saline-agglutinating antibodies in a titre of 1:1 on December 27, 1953. She had hydramnios. Her expected date of confinement was January 13, 1954, and she was advised to report on December 26, 1953, for premature induction of labour with possible exchange transfusion of

the baby. She was not seen until January 29, 1954, when she was admitted to the hospital in labour. The fœtal heart could not be heard nor the fœtal movements felt. At 9.45 p.m. the membranes ruptured, and it was noticed that the *liquor amnii* was heavily stained with meconium. At 5.20 a.m. the next day (January 30) she was delivered of a macerated fœtus. The placenta was removed manually, but was ragged and incomplete.

On January 31, at 3 a.m., she had a post-partum hæmorrhage; her blood pressure fell to 90 millimetres of mercury, systolic, and 40 millimetres, diastolic, and at 4.20 a.m. to 80 millimetres of mercury, systolic, and 50 millimetres, diastolic. Accordingly, a transfusion was commenced, and one bottle of "Plasmosan" (540 millilitres) and two bottles (1000 millilitres) of blood were transfused.

At 10 a.m. her blood pressure was 130 millimetres of mercury, systolic, and 80 millimetres, diastolic, but she looked ill. Her conjunctivæ were icteric, she was flushed and hot, her tongue was dry and coated, and her abdomen was distended. There were patches of bruising on the abdomen, and the urine was dark in colour. A cervical swab was examined, and encapsulated Gram-positive rods were found to be present in the smear; on culture *Cl. welchii* was isolated. The patient was transferred to the infectious block for further treatment. Her condition deteriorated rapidly, and at 3 p.m. the same day she was critically ill and it was feared that she would die. Her respirations were gasping, and she was jaundiced, with a rapid pulse and hot clammy skin. As she was now unable to take fluid by mouth, intravenous therapy was continued and she was given gas-gangrene antiserum, 60,000 units intravenously *statim* and 40,000 units every eight hours by intramuscular injection, and penicillin, 400,000 units every four hours. At 8.45 p.m. she looked brighter and did not have such severe respiratory distress; the hæmoglobin value was 68%. In anticipation of a fall in the hæmoglobin value she was given intravenously blood (500 millilitres), saline, 0·9% (250 millilitres) and dextrose, 5% (1000 millilitres) at 500 millilitres every four hours. The next day (February 1) her general condition had improved, but she was more jaundiced and her conjunctivæ were coppery in colour. She was able to take fluid by mouth, but had passed only 10 ounces of dark urine in the previous twenty-four hours. The blood pressure was 120 millimetres of mercury, systolic, and 75 millimetres, diastolic, her pulse rate was 80 per minute, and her temperature was 98° F. The hæmoglobin value was 38%. Two bottles of blood were transfused slowly, and the diagnosis of cortical necrosis was made.

On February 2 her general condition had further improved, although she was still obviously ill; her respirations were rapid. Her pulse rate was 80 per minute and the pulse was of good volume; the lung fields were clear. She was conscious and cooperative. The urine was small in amount and paler in colour, and contained "one-tenth" albumin.

On February 3 her condition had further improved; but, although she was cooperative, she was confused and said repeatedly that she would go home. Her blood pressure was 120 millimetres of mercury, systolic, and 75 millimetres, diastolic. She had two œdematous masses in the loins and pitting œdema of the sacrum. No signs of congestive cardiac failure were present. She was given peanut oil mixture, 35 ounces daily. Biochemical imbalance was shown by the following figures: the hæmoglobin value was 36%, the carbon-dioxide combining power was 20 volumes *per centum* (8·96 milliequivalents per litre), and the serum sodium content was 275 milligrammes per 100 millilitres (119 milliequivalents per litre). The patient was accordingly given repair solutions containing blood (1500 millilitres), 0·9 saline solution (250 millilitres), 2·5% saline solution (300 millilitres), and 4 molar sodium lactate solution (40 millilitres)—a total of 160 milliequivalents of sodium.

On February 4 at 10.15 a.m. she showed further slight improvement and was rational and cooperative. Her tongue was moist, and her pulse had a rate of 90 per minute and was of fair volume. Her colour had improved (the hæmoglobin value was 42%). She was given 1500 millilitres of blood very slowly; but when she was examined at 3 a.m. on February 5 her breathing appeared to be acidotic and she was therefore given sodium lactate, 40 millilitres of a 4 molar solution, in 500 millilitres of 0·9% saline by the drip apparatus, which was still running.

On February 5, at 9.30 a.m., her condition was greatly improved. The respiration rate had been reduced to 30 per minute, and her colour had improved. The peanut oil was continued. On February 6 her respirations numbered 42 per minute. Otherwise her condition was unchanged. She expressed a dislike for the peanut oil mixture. Because of the acidotic type of respiration, she was given by mouth an alkaline mixture containing 30 grains of sodium bicarbonate and 30 grains of sodium citrate six times daily in one ounce of water.

On February 7, apart from antagonistic behaviour to the nursing staff, her condition was unchanged. The lung fields were clear. The urine (one and a half ounces) contained "solid" albumin, and she had started to vomit and was unable to retain fluids taken by mouth. An unsuccessful attempt was made to pass a duodenal tube, and this was not repeated for fear of further distressing the patient.

On February 8, at 10 a.m., her general condition was good. She was cheerful and rested and was reading a paper. It was thought that her face looked swollen. The urine at this stage contained "solid" albumin. On February 9 her condition had not changed from the previous day, but her face looked a little swollen.

On the morning of February 10 her condition had deteriorated, she had signs of heart failure, her neck veins were distended—a sign which had not been noticed before—and her finger tips were cyanosed. She looked ill. Her blood pressure was 142 millimetres of mercury, systolic, and 76 millimetres, diastolic, her respirations numbered 50 per minute, and her skin was moist. She was conscious and cooperative and gave her consent to have her blood dialysed though she obviously did not understand what this meant and was too ill to try. Preparations for treatment by the artificial kidney were commenced, and meanwhile a course of "Digoxin" was begun, 0·25 milligramme being given intravenously every eight hours. The oral intake of fluid was limited to one and a half ounces per hour, and the alkaline mixture was discontinued.

Owing to unforeseen circumstances, the artificial kidney was not ready for use until late in the evening; it was decided, however, to perform the operation that night rather than to run the risk of losing the patient by further delaying it until the next morning.

At about 7.15 p.m. she was wheeled in her bed into the room occupied by the artificial kidney. In order not to frighten her, the machine with its blood-filled cylinders, rubber tubes and metal stands was hidden behind a screen except for the two rubber tubes with the glass cannulæ for insertion into artery and vein, which were lying on a small table. When the patient's bed had been put into the correct position, she was told that she would hear the constant noise of the machine during the operation, and it was then switched on and off a few times so as to accustom her to it. She was then given morphine sulphate (0·25 grain) subcutaneously, the right internal saphenous vein was exposed and a glass cannula inserted, and a slow drip administration of heparin solution, 25,000 units in 500 millilitres of 0·9% sodium chloride solution, was commenced. The radial artery was then carefully exposed; 25,000 units of heparin were then injected intravenously into the rubber tubing of the glass cannula in the leg vein, and after one minute the second glass cannula was inserted into the radial artery. As soon as blood appeared in the glass cylinder the artificial kidney motor was switched on and the dialysis commenced. It is worth mentioning at this stage that Kolff has pointed out that the utmost care should be taken when the radial artery is exposed to cause as little trauma as possible, because tiny branches of the radial artery can be a source of troublesome bleeding with the large doses of heparin used.

The motor of the artificial kidney was switched on at 8 p.m., and dialysis was continued without stopping for six and a quarter hours—that is, until 2.15 a.m. the next day. During the dialysis 50 litres of the bath water were run out and replaced on four occasions, as indicated in the chart (Figure III). As the bath originally contained 150 litres of fluid, it could be assumed that at least 75% of the bath water was thus replaced. As the bath water had not had any added calcium salt, it was feared that the dialysis might result in calcium deficiency, and accordingly, at 9.45 p.m., calcium gluconate (20 millilitres of a 10% solution) was injected intravenously slowly into the internal saphenous vein via the rubber tubing. This was followed immediately by vomiting and slowing of the patient's pulse, and the injection was consequently not repeated, as indicated in Table I. The serum calcium level did not fall.

Soon after the dialysis was started, the patient began to complain of itchiness, and at about 9 p.m. developed an urticarial rash, probably due to sensitivity to heparin, for which "Benadryl" (50 milligrammes) was administered. Apart from this and a slight shiver at about 9.30 p.m., her condition remained unchanged. Blood pressure, pulse and respiration readings were taken many times during the dialysis. Apart from slowing of the respiration, no change was recorded.

The patient slept most of the time during the dialysis and seemed a little brighter when the operation was completed; at that time five millilitres of protamine sulphate (1% solution) were injected intravenously and the cannulæ were removed, the wrist and ankle were bound up, a quarter of a grain of morphine sulphate was given subcutaneously, and the patient was wheeled back into her room. She slept well until about 8 a.m. and awoke looking and feeling much improved. Her face and hands were less swollen, although there was still swelling of her feet and sacrum. Her fingers were less cyanosed, she was rational and cooperative and more lively, she had ceased to vomit and had passed 940 millilitres of urine. Her blood pressure was still 160 millimetres of mercury, systolic, and 40 millimetres, diastolic, and her pulse rate 112 per minute; but her breathing was much easier.

Previous experience with this type of case has taught us that the danger period has not been passed until many days after the diuresis sets in. Accordingly, fluids were carefully regulated. Biochemical estimations were made, and "Digoxin" (0·25 milligramme twice a day) was administered intravenously for two days. As her temperature was still raised and the penicillin appeared to be without effect, it was discontinued and the oral administration of "Aureomycin" (250 milligrammes daily in two doses) was commenced. This, too, was without effect in controlling the temperature, but the general condition of the patient continued to improve. On February 13 she developed a macular rash, which was confluent on her back, and petechiæ on the abdomen. At the same time, she developed a left pleural friction rub; by this time the "Digoxin" therapy had been discontinued. The rash did not appear to cause her any discomfort and faded in forty-eight hours. The œdema of her feet and sacrum had continued to diminish and had disappeared by the morning of February 14. The veins of her hands were less prominent now, and in place of the œdema there were small wrinkles in the skin of the hands. The pleural friction rub lasted about five days; an X-ray film of the chest showed no abnormality. She now entered a period when it appeared that the voluminous diuresis would result in dehydration. Her face was small and pinched on February 15, and as she was unable to drink sufficient fluids she was given intravenously blood (500 millilitres) and 0·9% sodium chloride solution (300 millilitres) on February 15, and blood (1000 millilitres), 5% glucose solution in 0·9% sodium chloride solution (1300 millilitres), and 10% potassium chloride solution (60 millilitres—80·4 milliequivalents) on February 16. No diuresis followed this infusion (Figures I and II).

TABLE I.

Serum Protein, Serum Calcium and Hæmoglobin Readings During Dialysis.

Time.	Calcium. (Milligrammes per 100 Millilitres of Serum.)		Hæmoglobin Value. (*Per Centum.*)[1]		Protein. (Grammes per 100 Millilitres.)	
	Arm.	Leg.	Arm.	Leg.	Arm.	Leg.
8.30 p.m. ..	12·7	9·0	75	74	6·5	6·3
9.15 p.m. ..	12·7	9·7	76	75	6·5	6·5
10.00 p.m. ..	12·5	10·6	76	75	6·5	6·5
11.30 p.m. ..	11·6	—	75	75	6·2	6·5
12.45 a.m. ..	10·8	12·0	74	75	6·2	6·0
2.15 a.m. ..	11·0	10·6	72	76	6·3	6·1

[1] 100% = 15 grammes *per centum*.

She was much improved after this and was able to sit up in a chair on February 17. She continued to improve; but as the blood pressure and serum potassium level were still low, she was given intravenously on February 25 blood (1000 millilitres), 5% glucose solution in 0·9% sodium chloride solution (2000 millilitres), and 20 millilitres of a 10% solution of potassium chloride (26·8 milliequivalents of potassium). This infusion resulted in diuresis and an increased excretion of sodium, chloride and potassium.

On March 1 a piece of placenta was expelled. This was not offensive, but served to explain the elevated temperature, as she had no lochia at the time. Nevertheless, she continued to improve and on March 3 she was able to walk with assistance. On March 5 the vaginal discharge was slight and yellowish. The uterus was enlarged to the size of a six weeks' pregnancy, the fornices were not tender, the os admitted one finger. A cervical swab was taken, and a course of chloramphenicol (500 milligrammes every eight hours) and penicillin (800,000 units three times a day for twenty-four hours) was begun; her uterus was curetted the next day (March 6). A large amount of yellowish necrotic material was removed from all areas of the uterine wall, which appeared like necrotic endometrium. The uterus was firm and mobile, the fornices were clear, and there were no masses or induration in the pelvis. Hæmorrhage was slight. The temperature settled to normal after the curettage. The vaginal discharge ceased, but the blood pressure remained low. Nevertheless, her general condition continued to improve. She had a good appetite and was walking about. On March 16 she was discharged from hospital with a blood pressure of 95 millimetres of mercury, systolic, and 60 millimetres, diastolic. She was examined as an out-patient on April 10, when the blood pressure was 100 millimetres of mercury, systolic, and 60 millimetres, diastolic, and on May 10, when it was 100 millimetres of mercury, systolic, and 60 millimetres, diastolic. She has been well since, and has put on a stone in weight since her discharge from hospital.

Discussion.

Anuria or oliguria associated with infection is accompanied by a high mortality in spite of chemotherapy. The maintenance of correct water balance and electrolytic equilibrium in these patients is difficult, and they require to be constantly visited and reassessed. In the case of Mrs. B., the massive hæmolysis which required seven bottles of blood to maintain the hæmoglobin level at 70% (10·5 grammes *per centum*) was particularly dangerous because of the liberation of potassium ions, sulphate and phosphates which could not be excreted. It is therefore surprising that the serum potassium level did not rise higher than it did (22·5 milligrammes per 100 millilitres on February 10—that is, after ten days of oliguria). An electrocardiogram was taken on February 16, February 17 and February 25. No characteristic signs could be demonstrated. She probably lost some potassium in the small amount of daily vomitus, but it is unlikely that the serum potassium level was maintained near normal figures in this way.

Because of the very low carbon dioxide combining power during the early days of treatment, she was given sodium lactate as a concentrated (4 molar) solution. The carbon dioxide level was raised, but never reached normal figures, and the improvement was noticed clinically rather than demonstrable biochemically. The administration of sodium ions must undoubtedly have helped to expand the extracellular space and increase œdema, which was present in the dependent parts. She was nursed in a propped-up position, and the œdema was present in the buttocks and loins and the lower extremities. At the time it seemed wise to attempt to maintain the carbon dioxide combining power at a reasonable level by sodium lactate and alkaline mixtures in spite of the œdema. It is unlikely that the œdema itself contributed much to her cardiac failure later, as the severe infection she had had to combat and the ten days of uræmia following this were sufficient to account for it.

Electrolytic Changes During Dialysis.

Serial blood specimens were taken from the arterial blood which was flowing into the artificial kidney; these specimens have all been designated "arm" specimens in Figure III, and examination of them indicated changes which took place in the patient.

Simultaneously with each of these specimens, a specimen of blood was taken from the blood flowing into the saphenous vein of the patient after passage through the artificial kidney. These specimens are designated "leg" specimens, and examination of them indicated changes which had taken place in the blood after its passage through the artificial kidney. All specimens were actually taken from a side arm in the tubing, but not from the patient herself, and were analysed later. The serum sodium, potassium and chloride values are given in milliequivalents per litre. The blood urea and blood sugar findings are given as milligrammes per 100 millilitres of blood (Figure III).

The serum potassium level in the arm (given at the head of the graph, Figure III, for convenience) remained stationary during dialysis. When dialysis first commenced, the bath water did not contain any potassium

chloride. At the time of changing the bath water—indicated by arrows at the foot of Figure III—potassium chloride was added to the bath water to prevent undue loss of potassium ions. The initial lower serum potassium values in the leg specimens are probably the result of loss of potassium ions by dialysis. The final values for the leg specimens, which are slightly higher than those for the arm specimens, probably resulted from hæmolysis of

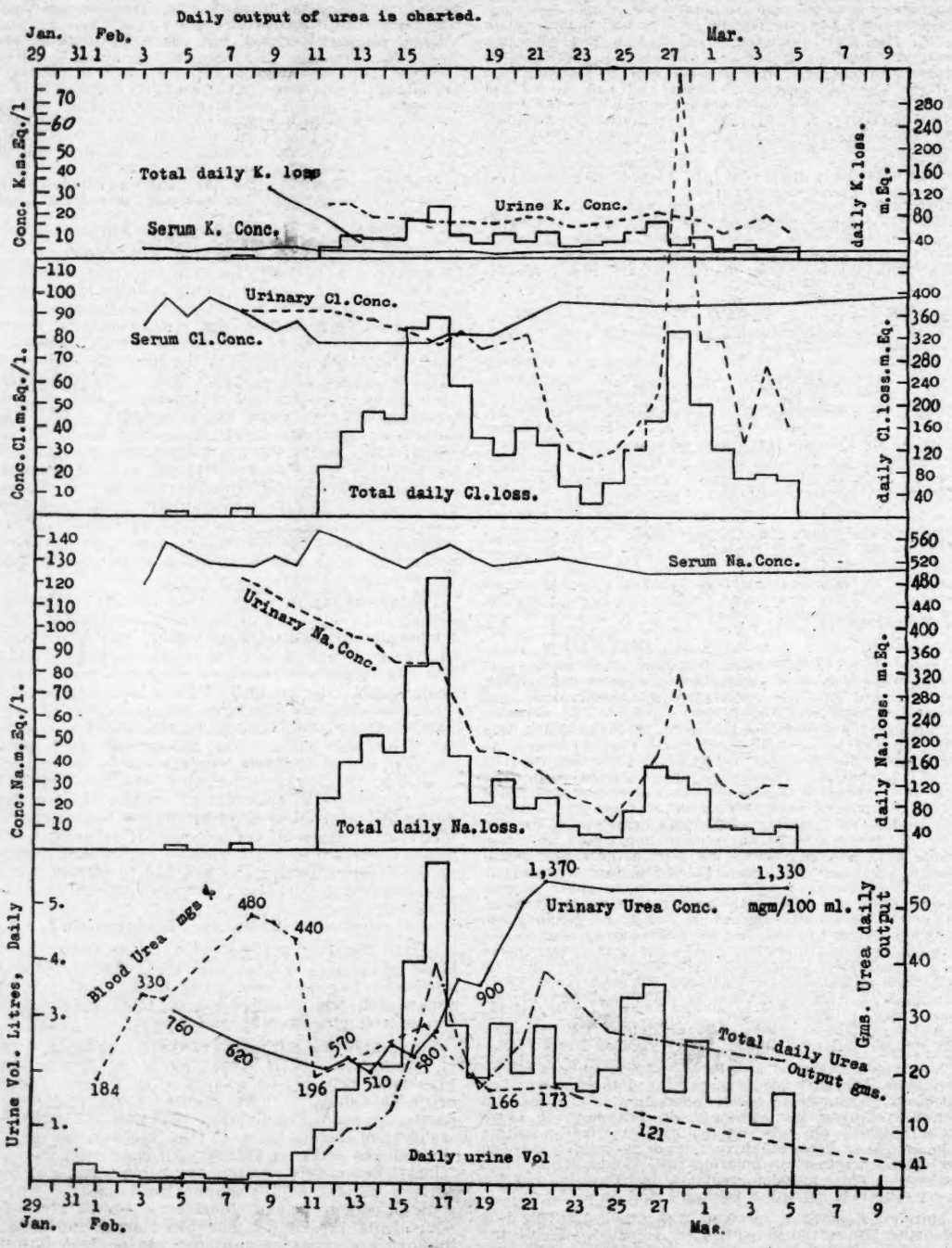

FIGURE I.

The total daily urinary output of water in millilitres per twenty-four hours, and of sodium, potassium and chloride in milliequivalents per twenty-four hours, is shown as a block above the base line. The urinary and serum concentrations of sodium, potassium and chloride are shown as a graph. The blood urea and urinary urea estimations in grammes per 100 millilitres are shown as a graph; each is drawn to a different scale in order to achieve proximity, but the values are noted on the graph. The daily output of urea is shown.

blood due to drying up in the side arm from which the specimens were taken or to mechanical action in the artificial kidney, as the concentration of potassium in the bath water was lower than this.

The blood urea level in the leg and arm specimens showed a continuous fall. The first leg specimen, taken half an hour after the kidney had been in action, contained only 124 milligrammes of urea per 100 millilitres of blood 100 millilitres of blood. The values for the arm specimen also rose and followed those for the leg specimens, just as the fall in blood urea level in the arm specimens had followed the fall in blood urea level in leg specimens. The rise in blood sugar content above normal levels is not in itself harmful. The provision of calories is beneficial, and the blood sugar content could be expected to fall to normal levels in approximately five hours in a non-diabetic. Kolff

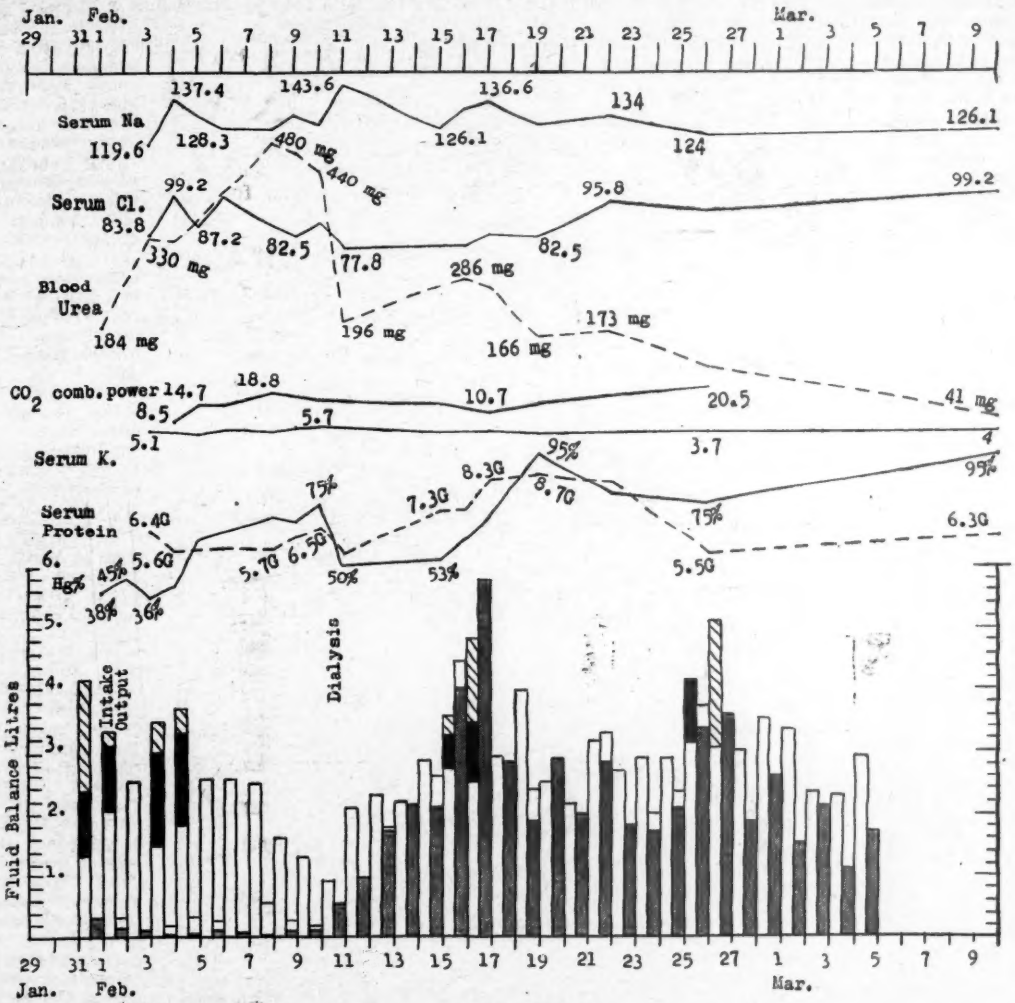

FIGURE II.

The values for serum sodium, potassium and chloride contents, and for the carbon dioxide combining power, are given in milliequivalents per litre. The blood urea content is given in milligrammes per 100 millilitres of blood. The serum protein content is given in grammes per 100 millilitres of serum. The hæmoglobin value is expressed as a percentage, 100% being equal to 15 grammes per 100 millilitres of blood. In the "intake" columns, the oral fluid intake is shown by an open column, the blood by a solid column and the total intravenous saline intake by a cross-hatched column; on the "output" side, urine is shown by a lightly shaded column and vomitus by an open column.

against the initial level of 440 milligrammes, and fell continuously thereafter. The values for the arm specimens fell more gradually, as was to be expected, but had reached the low figure of 196 milligrammes of urea per 100 millilitres of blood after six and a quarter hours of dialysis.

The blood sugar values for the arm and leg specimens are all high and are just the reverse of those obtained for urea. The first leg specimen had a concentration of 392 milligrammes of glucose per 100 millilitres of blood, which rose continuously to a level of 460 milligrammes per

has shown that œdema may be lessened by increasing the sugar (dextrose) concentration of the bath water; however, it did not appear wise to attempt too much, and it must be assumed that the patient's œdema was not altered much, although her face and hands were less swollen the next day.

The serum sodium and serum chloride findings were not so constant as the other findings. The bath water had a concentration of 0·6 gramme of sodium chloride per 100 millilitres—that is, 102·6 millimols per litre of sodium and

chloride each—and it might be expected that the serum chloride would therefore be constant in both the arm and leg specimens. On the other hand, the artificial kidney had not been provided with a hood to prevent evaporation of water, which would tend to make the solution more concentrated. The passage of sodium bicarbonate from the blood to the bath water was prevented by the sodium bicarbonate in the bath water (0·2 gramme of sodium bicarbonate per 100 millilitres—that is, 23·8 milli-equivalents per litre of sodium and bicarbonate each), so that no appreciable change should occur in these ions.

As the serum sodium and serum chloride values for the leg specimens were found to be higher than those for the arm at all times, it must be assumed that sodium chloride was dialysed into the patient and that irregularities in the graph are due to technical error. The volume of blood which had passed through the artificial kidney was measured at many stages of the proceedings and is recorded in Figure III. As can be seen, the flow was kept fairly constant. The total amount of blood which had been dialysed after six and a quarter hours was 46,200 millilitres.

The hæmoglobin and serum protein levels remained constant in the arm and leg specimens during dialysis, which suggested that the water balance of the patient was not influenced appreciably. The hæmoglobin value dropped some hours later, probably because of trauma to red cells in the artificial kidney.

The serum calcium levels in both arm and leg specimens remained constant. Calcium chloride was not added to the bath water because calcium carbonate would be precipitated. The calcium gluconate given intravenously to the patient resulted in a slowing of the pulse and a feeling of nausea, and was not repeated.

The blood pressure and pulse remained normal and were unchanged during dialysis.

The urine was examined daily, and the total daily loss of sodium potassium and chloride in millimols and of urea in milligrammes was estimated. These losses are shown graphically in Figure I, together with the serum and urinary concentration of these substances and the daily output of urine. An inspection of this graph reveals the following facts.

1. From January 31 to February 10 the urine output was very low and the blood urea level rose; the slight apparent drop in blood urea level on February 4 and February 10 is probably due to technical error in estimation. A very dramatic drop in the blood urea level followed the dialysis. It then rose again until February 16, and after this it fell gradually to normal figures.

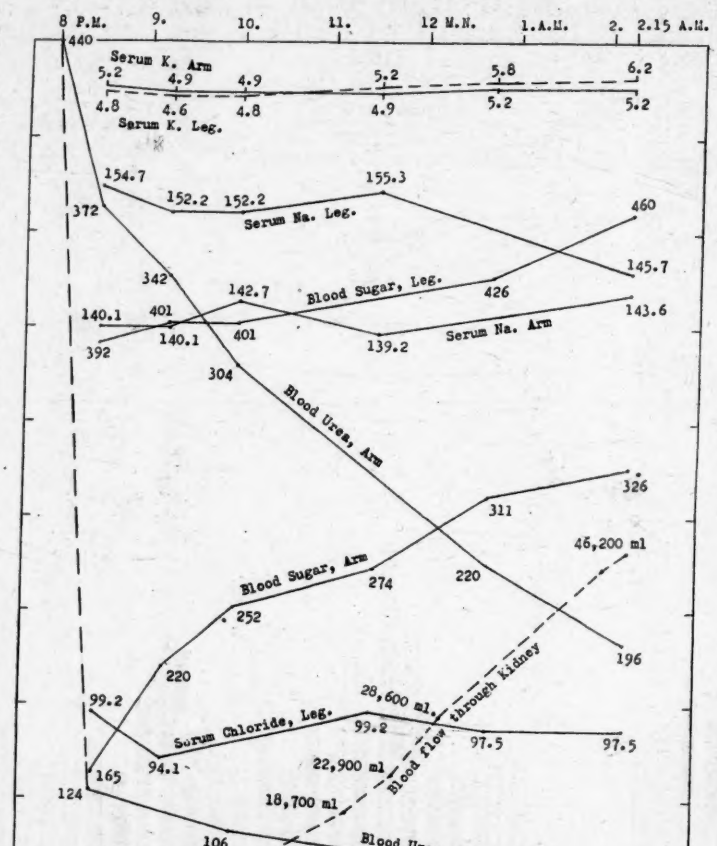

FIGURE III.

The serum sodium, potassium and chloride contents are given in milli-equivalents per litre. The blood sugar and blood urea contents are given in milligrammes per 100 millilitres of blood.

2. The urinary urea concentration stayed at a fairly constant level until about February 16, and then rose.

3. The total urea output was at first (until February 17) dependent on the total amount of urine voided. Subsequently it was excreted in greater proportion, and this in spite of the fact that the blood urea level was much lower than previously. In other words, the initial urine resembled a glomerular filtrate and subsequently become more like normal urine.

4. During the stage of diuresis—that is, until about February 18—there was a great loss of sodium, chloride and potassium ions, which was reflected in the serum sodium, chloride and potassium levels, except when these were modified as a result of intravenous therapy (Figure II). The loss of these ions was not strictly proportional to the loss of water in the urine, because the urinary concentration of sodium, and to a lesser extent potassium and chloride as well, fell during the period of diuresis. This suggests that there was an early renal attempt to conserve sodium and to a lesser extent the other ions as well.

5. The power to conserve sodium seems to have been regained first, then the power to conserve water and to excrete urea in high concentration.

6. The increased loss of sodium, chloride and potassium ions following transfusion and infusion on March 25 and March 26 was achieved by the passing of more concentrated urine with respect to these ions and may be taken as a further indication of renal improvement. The specific gravity of the urine was 1010 daily till March 5. The urine always contained a trace of albumin.

In Figure II, the blood biochemical findings have been recorded, with fluid intake from all sources and fluid loss

from all sources. The low hæmoglobin values caused by the initial hæmorrhages are noteworthy. There was a fall in hæmoglobin and serum protein levels following dialysis, and a rise in hæmoglobin and serum protein levels during the period of diuresis caused by extracellular dehydration. The rise in the hæmoglobin level was also partly due to transfusion, and after this rise there was a fall as the power to conserve sodium chloride and water was regained. The hæmoglobin and serum protein levels then rose with further transfusion and increased food and hæmatinics to an expected level. The fall in hæmoglobin value and serum protein concentration following dialysis might suggest that the patient had been further hydrated during the dialysis. In actual fact, her face and hands looked less swollen after dialysis; her hands during dialysis were in the dependent position, as she was nursed propped up. In the absence of total body weight determination, it cannot be stated with certainty whether there had been a total loss or gain of body water, and further cases will therefore have to be studied to determine whether the hæmoglobin and serum protein values fall regularly. Part at least of the drop in hæmoglobin value could result from hæmolysis of blood following mechanical action in the artificial kidney.

Diet.

The diet given to the patient was as follows.

1. When she first became ill on January 31, she was able to take only clear fluids, chiefly water and glucose water.

2. On February 3, when the infection had been controlled, she was given peanut oil emulsion, 35 ounces daily,[1] which she took without complaint until February 6. After this, however, she was given only 18 ounces of the mixture daily with some glucose water. She was also given a very small amount of orange juice and jelly, in order to relieve the monotony of the diet, though not in sufficient amounts to be harmful because of its potassium content.

3. On February 8 the total intake of fluids was curtailed on account of the œdema.

4. When the diuresis had commenced she was allowed to have, in addition to the peanut oil and glucose water, tea, fruit juices, fruits, and some ice cream and a little bread, butter and jam.

5. She was not allowed to have meat, eggs or milk in large amounts until February 24, when the fall in blood urea level appeared to be progressive; from this time the diet was increased until on March 5 she was taking a normal diet which she ate with relish.

[1] Peanut oil, three ounces; glucose liquid, 14 ounces; gum acacia, *quantum sufficit*; water to 35 ounces.

Description of the Artificial Kidney.

It was decided at the beginning to make use of only such material as was readily available in order to minimize the cost and facilitate manufacture. Accordingly, an adult plastic bath was fitted into a ward trolley, and the rest of the artificial kidney built into this (Figure IV). The advantage of this was that the bath could be easily cut and fitted. The bath being large minimized the danger of the dialysing fluid becoming too hot or cold; it also made it unnecessary to change the bath water frequently, because the huge volume (150 litres) would form a veritable sea in which the concentration of toxic products from the dialysate would not rise and a good diffusion gradient would be assured. The trolley was a firm structure onto which the rest of the equipment could be fixed, and it was movable from place to place.

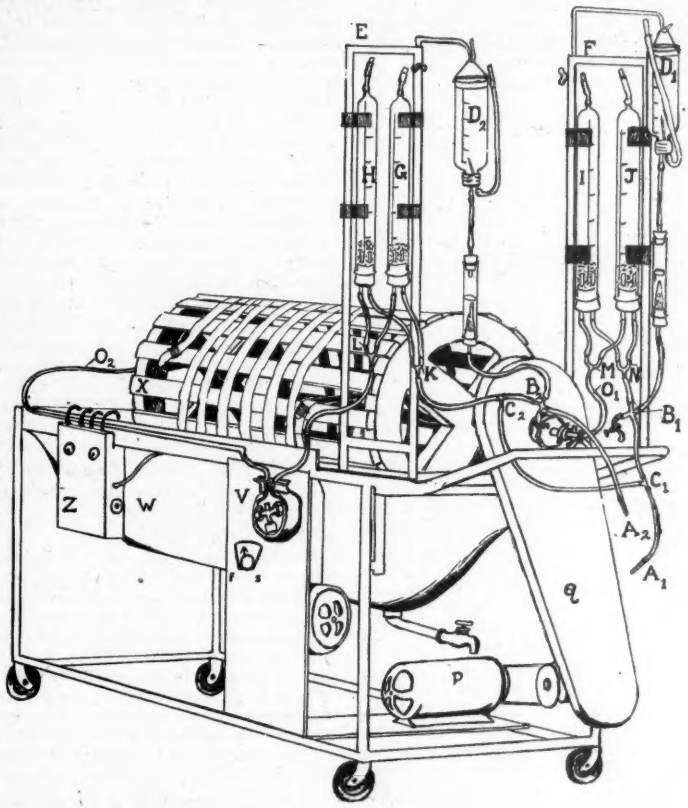

FIGURE IV.

Figure IV is a semi-diagrammatic drawing of the machine.

W is the plastic bath fitted into the trolley.

X is the hollow cylinder made of wooden slats of beech wood; it is 52 inches long and 18 inches in diameter. The hollow cylinder is fitted at either end by metal bearings to the steel frame of the trolley and is rotated by a 0·5 horse-power electric motor, P, situated under the bath. The lower one-third of the hollow cylinder dips into the dialysing fluid in the bath during the working of the machine. The "Cellophane" dialysing tubing, three-quarters of an inch in diameter, 120 feet in length, is wound around the hollow cylinder.

Z is a thermostatically controlled element for maintaining the temperature of the bath water at blood heat.

E and F are two detachable stands made of chromium-plated tubular brass. These stands fit into slots on the frame of the trolley, and support the 500 millilitre graduated cylinders G, H, I and J. Each stand has an adjustable arm for the support of the 500 millilitre transfusion bottles D_1 and D_2, each with their transfusion sets.

During sterilization each stand with its graduated flasks and all connexions from one side of the artificial kidney (except the transfusion sets) are made into one package and sterilized with the parts assembled, so that during the setting up of the machine it is necessary only to fix the stands into the frame of the trolley and straighten the rubber tubing into place. There are thus two packages, one labelled "arm", which contains all the apparatus for

conveying blood from patient to machine, and another labelled "leg", which contains all apparatus for conveying blood from machine to patient.

V is an electrically controlled Julian Smith pump with adjustable speeds of revolution.

B_1 and B_2 are glass T pieces.

K, L, M and N are glass Y pieces.

The mouth of each of the four 500 millilitre graduated flasks is closed by means of a well-fitting rubber bung held firmly in place by a metal clamp (not shown). Each bung is traversed by two glass pieces; the upper ends are fish tailed, the lower ends are connected by means of rubber tubing to the glass Y pieces K, L, M and N. Each 500 millilitre graduated flask contains a handful of glass beads which cover the fish tails and act as a filter. The upper end of each flask is closed by means of a simple air filter as in the transfusion sets. The rubber tubing used was three-sixteenths of an inch by one-sixteenth of an inch transfusion tubing.

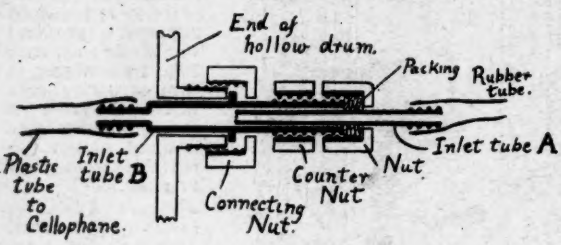

FIGURE V.

The manner in which the blood passes from the tube O_1 to the "Cellophane" tube and from the "Cellophane" tube to the tube O_2 is shown diagrammatically in Figure V. O_1 and O_2 remain stationary, while the "Cellophane" tube rotates with the hollow drum, and for this reason a well fitting connexion is essential. By reference to Figure V, it will be seen that the blood passes from the rubber tube (O_1) into the stainless steel tube A, and from here through the inlet tube B to a wide-bore plastic tube about 12 inches long, which extends as far as the slats of the hollow drum and is tied there with tape. The "Cellophane" tube fits over the plastic tube and is tied on with tape. The nut and counter-nut hold the packing firmly in place and prevent leakage. The connecting nut holds tube B firmly on to the hollow drum. In this way, the blood is led from a stationary tube to one that is rotating, as the hollow drum, tube B and the plastic tube are rotated by the motor around the axis of the drum.

When the machine is to be used, three packets of salt, each containing dextrose (500 grammes), sodium chloride (300 grammes), sodium bicarbonate (100 grammes) and potassium chloride (20 grammes), are dissolved in a known volume of tap water, and the solution is poured into the bath. Tap water is then added to make a total of 150 litres; this gives a total concentration of 1·0 gramme *per centum* of dextrose, 0·6 gramme *per centum* of sodium chloride, 0·2 gramme *per centum* of sodium bicarbonate, and 0·04 gramme *per centum* of potassium chloride. The solution therefore has a total concentration of 126·39 milliequivalents per litre of sodium, 107·96 milliequivalents per litre of chloride, 23·79 milliequivalents per litre of HCO_3, 5·36 milliequivalents per litre of potassium, and 55·5 millimols per litre of dextrose. The solution thus has a total osmotic pressure of 319 milliosmols. For the sake of convenience, the potassium chloride is kept in separate packets and added to the water only when required. The heating element is then turned on and the two stands E and F with their assembled apparatus are fixed in place. The "Cellophane" is then wound tightly onto the drum and tied to the plastic tubes. The whole apparatus is then flushed through with about 10 litres of sterile 0·9% sodium chloride solution, each bottle used being put up at D_1; the last bottle contains 25,000 units of heparin. Finally, three bottles of blood are run into the machine, so that all the rubber tubes and glass connexions and the "Cellophane" tubing are filled with blood which has displaced the sodium chloride solution. Only one graduated cylinder on each side of the machine is filled with blood, the other being kept empty; this is done by applying Spencer Wells forceps to the rubber tubing above K, L, M and N wherever required. "Soluvac" tubing clamps take too long to manipulate, and therefore Spencer Wells forceps are preferred.

It is necessary to have at least two operators to work the machine. When the patient is heparinized and the cannulæ A_1 and A_2 are inserted into the radial artery and saphenous vein respectively, blood flows into the apparatus along A_1, B_1 and N. From here, it can be diverted into either graduated flask I or J, whichever is empty, by clamping off the alternative route with a Spencer Wells forceps. At the same time, the blood in the filled flask J or I is allowed to flow into the artificial kidney via M and O_1 by suitable manipulation of the Spencer Wells forceps. Blood is propelled along in the "Cellophane" tube by the rotations of the hollow cylinder, the tube acting like an Archimedes pump. During its passage through the "Cellophane" the blood is dialysed. The action of the Julian Smith pump propels blood from O_2 through L into either flask G or H (whichever is empty), while at the same time the filled flask H or G empties by gravity into the vein via K, B_2 and A_2. One operator on each side of the machine is required to manipulate the Spencer Wells forceps and to keep a running total of the amount of blood passing through his cylinders, and both work synchronously so that blood leaves and enters the patient at the same rate; as a result the blood volume of the patient is maintained constant within narrow limits, any physiological trauma being thus prevented.

It must not be forgotten that patients who receive extra-corporeal dialysis are sick people and stand changes in blood volume poorly.

Commercial models of the Kolff artificial kidney have a flowmeter to measure the rate of blood flow through the kidney; as the rate of blood flow tends to vary, this is an inaccurate means of estimating blood flow. In addition to this, commercial models have no means of controlling the amount of blood that flows out of and into the patient, and the artificial kidney built here is unique in this respect. The manipulation of the forceps, though time-consuming, is not tedious, and the complete control of the blood volume of the patient greatly lessens any danger of either exsanguination or cardiac overloading and is well worth the effort.

When the machine is ready for use, the cannula A_2 is inserted into the internal saphenous vein, and a slow drip of 25,000 units of heparin in saline is allowed to flow in from D_2. The radial artery is then exposed with as little trauma as possible, and then 25,000 units of heparin are injected into the internal saphenous vein via the rubber tubing. After a delay of one minute the cannula A_1 is inserted into the radial artery; and as the blood flows out, the motor of the machine is switched on and dialysis proceeds. The 25,000 units of heparin in saline in D_2 are allowed to run in during the dialysis in about six hours.

Blood samples are taken at B_1 and B_2 via the side arm of the glass X pieces.

C_1 C_2 is a rubber tube with two T pieces of glass at either end fixed in position as shown. This forms a short circuit between artery and vein. Normally this tube is clamped off. However, if any defect should occur in the machine, the blood may be diverted through this circuit until the defect is rectified.

P is a 0·5 horse-power motor situated under the bath, which rotates the hollow cylinder through a reduction gauge. The driving belts are shielded from splash by the hood (Q).

Acknowledgements.

I wish to thank Dr. D. L. Wrench and Dr. B. H. Gutteridge for assistance in the setting up and working of the machine, and Dr. D. A. Henderson for advice during treatment of the patient. I also wish to thank Mr. H. Lloyd, chief electrician, Brisbane General Hospital, and his staff for their ingenuity and cooperation in building the artificial kidney in the electricians' department of the Brisbane Hospital. Finally I should like to thank Dr. A. E. Shaw, Director of the Red Cross Blood Transfusion Service (Queensland Branch) for the loan of relevant literature.

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A, B, C ELECTROCARDIOGRAPHIC DIAGNOSIS OF INTERATRIAL DEFECT WITH A NOTE ON THE BRUIT AND THE FINDINGS IN INTERVENTRICULAR DEFECT.

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THE diagnosis of interatrial defect is usually simple, but not always. In straightforward cases the findings are as follows: a "systolic" bruit in the second or third left intercostal space close to the sternum, commonly associated with a widely split second sound at the base, electrocardiographic findings stated to indicate "partial bundle block" in 95% of cases, congested pulmonary fields, prominent pulmonary artery, hilar dance, right atrial enlargement, a small aorta, and possibly congenital anomalies. Catheter studies yield a high oxygen content of right atrial blood or movement of the catheter occurs directly from the right to the left atrium (seen radiologically). However, in some cases the diagnosis is not always made so readily. In this paper evidence is produced of a more generally characteristic (and probably diagnostic) feature of interatrial defect which is obtained in the electrocardiogram with the use of leads A, B and C. (Trethewie, 1951, 1953). Further, a note is presented on the feature of the bruit itself.

Method.

Electrocardiograms were prepared on a Both machine, and phonocardiograms, with electrocardiographic timing, on a cathode ray oscillograph supplied by Mr. D. Dewhurst. The microphone had a diameter of 5·5 centimetres, and was held in place by the hand. The amplification was linear.

Electrocardiographic Findings in Interatrial Defect.

The following three patients illustrate the electrocardiographic findings in patients with obvious interatrial defects.

The tracing of patient P1 is recorded in Figure I. It is noted that the *R* wave in lead A has a double contour. Commonly a step is seen on this wave in interatrial defect (Trethewie, 1953); but here (Figure I) the step wave is of greater potential than that of the succeeding *R* wave. The *T* wave is reduced in amplitude and in leads B and C inverted to biphasic. There is also a *Q* wave in lead B. This indicates a degree of *cor pulmonale*, a common enough sequel of interatrial defect. It is the step attached to the *R* wave (lead A) which is the feature indicating interatrial defect.

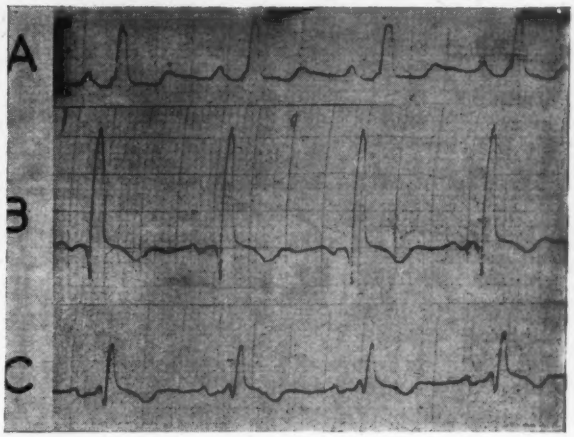

FIGURE I.

The electrocardiogram of leads A, B and C in patient P1 with interatrial defect. Note the double contour *R* wave in lead A, with the initial wave potential (0·8 millivolt) of greater voltage than the succeeding *R* wave. Note the *Q* wave in lead B and inverted and biphasic *T* waves indicative of considerable right cardiac strain.

Patient P2, a female, aged forty-seven years, had a readily recognizable interatrial defect. A systolic murmur was heard in the third left intercostal space with a loud diastolic bruit down the left border of the sternum. The phonocardiogram of this bruit is presented later (Figure VIII). Fluoroscopic examination revealed an enlarged heart with hilar dance and a huge pulmonary artery. The X-ray film showed the heart shadow to be considerably enlarged and the right ventricle to be big. There was a huge pulmonary shadow with a small aortic knob, and the pulmonary vessels were very large. The electrocardiogram (Figure II) shows in lead A a step on the *R* wave of 0·7 millivolt. The *R* waves are prominent, indicating preponderance of the right side of the heart.

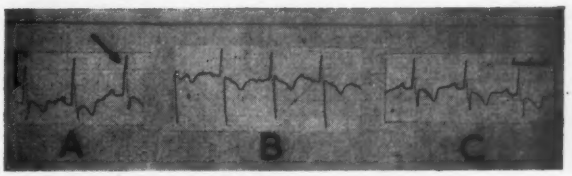

FIGURE II.

The electrocardiogram of leads A, B and C in patient P2 with interatrial defect. Left hand panel: note the "step-wave" with shouldering effect on the *R* wave marked with the arrow (this has a potential of 0·8 millivolt), also inverted *T* wave (and prominent *R*). Middle panel: *R* wave nearly equal in potential to the *S* wave. Right hand panel: prominent *R* wave, inverted *T* wave.

This is a classical tracing of interatrial defect, and it is noted that the *T* waves are inverted; this is probably indicative of right cardiac strain, though only moderate in degree in view of the absence of a *Q* wave in lead B.

Patient P3, a female, aged twenty-two years, also had typical signs of interatrial defect—namely, a typical quality systolic bruit in the third left intercostal space adjacent to the sternum—and X-ray examination revealed right cardiac enlargement, a large pulmonary artery, hilar dance and

[1] Technical assistance for this work was aided by a grant from the National Health and Medical Research Council, Australia.

pulmonary plethora. The electrocardiogram (Figure III) shows a step on the *R* wave of greater potential than that of the immediately following *R* wave (marked with an arrow), with a large *Q* wave in lead B and inverted and biphasic *T* waves. The findings, as with P1, indicate interatrial defect with *cor pulmonale*. There is also a small step wave on the *R* in lead C, indicating either a large interatrial defect or axis rotation with altered heart contour.

The following case illustrates obvious A, B, C lead electrocardiographic findings in a case in which the clinical diagnosis of interatrial defect was not so obvious.

Patient P4, a female, aged forty-nine years, was recognised as having a congenital heart lesion with a systolic

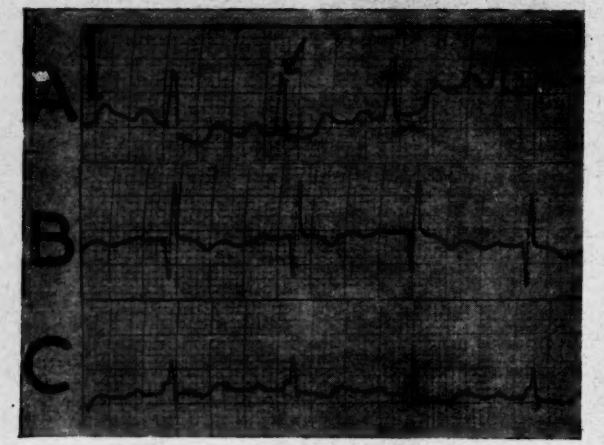

FIGURE III.

The electrocardiogram of leads A, B and C in patient P3 with interatrial defect. Note the double *R* wave formation in lead A marked with the arrow, also the large *Q* wave in lead B with biphasic *T* waves in lead C indicative of right cardiac strain.

bruit to the left of the sternum and a standard electrocardiogram consistent with the diagnosis of pulmonary stenosis. To me the bruit was of the characteristic "interatrial" type (*vide infra*) and maximal in the third left intercostal space. An electrocardiogram (Figure IV) was then taken, which showed a much greater initial element to the *R* wave than in the preceding case, a very large *P* wave in lead A and a *Q* wave of 1·5 millivolts in lead B. The *T* wave was inverted in all leads. The diagnosis of interatrial defect was confirmed, and *cor pulmonale* was present. In addition a step of 0·7 millivolt was noted on the *R* wave in lead C. This would indicate that the defect produces alteration of the action current in the antero-posterior direction. This is noted in minor form in the previous case (P3—Figure III). Further investigation by catheterization showed that nearly fully oxygenated blood (95%) was obtainable from a catheter placed in the right atrium. Polycythæmia was present (8,200,000 erythrocytes per cubic millimetre). X-ray examination showed the heart to be considerably enlarged, with prominence of the pulmonary artery and considerable enlargement of the heart to the right. The aortic knob was reduced, the right ventricle was enlarged, pulmonary plethora was evident and X-ray examination with a barium bolus revealed no abnormality.

The following case illustrates the electrocardiographic findings when there is an associated cardiac lesion in addition to interatrial defect.

Patient P5, a female, aged fifty-nine years, was considered to have an interatrial defect. She had been breathless on exertion for as long as she could remember, and on examination was slightly cyanosed; her blood pressure was 180 millimetres of mercury, systolic, and 110 millimetres, diastolic, and the radial vessels were thickened. A systolic bruit was heard, maximal in the third and fourth left intercostal spaces near the sternum. Fluoroscopic examination revealed a very large heart with regular slow pulsation of the left border and a large pulmonary artery. The right auricle had a different rhythm. Hilar dance was present. The X-ray film showed an extremely large heart with a large pulmonary artery and moderate pulmonary plethora. The tracing (Figure V, October 5, 1952) showed in lead A an extra wave form of low voltage immediately preceding the *R* wave, but the *R* wave had only a little greater potential. The step appearance was lost, probably because of the very low potential and time duration of both waves. A notable *Q* wave in lead B with flat to biphasic *T* waves indicated *cor pulmonale*. On August 7, 1953, this patient had a sudden attack of "terrific" pain in the chest, left arm and neck, which lasted for six hours. This had been preceded by attacks (for two weeks) of submammary pain relieved by trinitrin. A standard leads electrocardiogram on September 29, 1953, revealed the presence of a complete right bundle (new terminology) branch block. The patient had apparently suffered a coronary occlusion, in view of the history.

For comparison with the typical electrocardiographic findings in interatrial defect, the typical appearances of the electrocardiogram in uncomplicated pulmonary stenosis are shown in Figure VI, a tracing taken from patient P9. Prominent *R* waves (two millivolts) are noted. There is no step-wave formation on the *R* wave. The *T* waves are inverted in all leads, and there are no *S* waves. This is a classical tracing of pure uncomplicated pulmonary stenosis, and the absence of *cor pulmonale* is indicated by the absence of a *Q* wave in lead B.

Phonocardiographic Findings in Interatrial Defect.

The bruit is usually described as a systolic bruit heard just to the left of the sternum in the second or third left intercostal space. A basal diastolic murmur (50% of cases) is also described (Evans, 1948; Paul Wood, 1952; Levine, 1951), said to be due to functional pulmonary incompetence. The bruit has a characteristic quality. It has a "rrupp" accentuation in it difficult to describe, and possibly due to the early stage in systole at which it occurs and to a rapid *crescendo* quality with more gradual *diminuendo*. In timing, if the bruit was caused by movement of blood between the atria, one might expect to hear a presystolic element corresponding to the "a" wave in time. This is rarely heard, and is usually when present

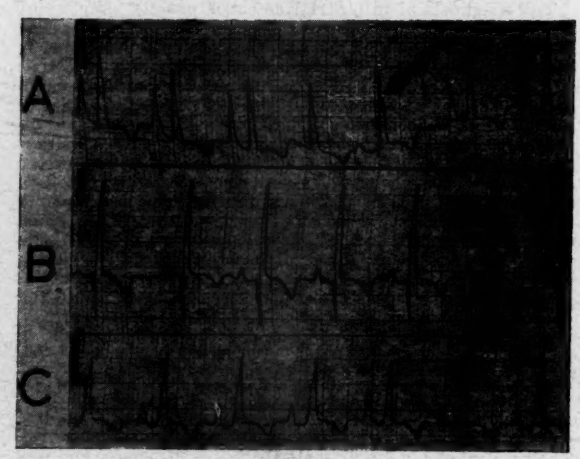

FIGURE IV.

The electrocardiogram of leads A, B and C in patient P4. Lead A: note shouldering effect on the *R* wave marked with arrow, initial wave (1·4 millivolts) of greater potential than succeeding *R* wave. Very prominent *P* wave, *S* wave negligible. Lead B: marked *Q* wave, large *R* wave (3·8 millivolts), no *S* wave. Lead C: prominent *R* wave with shouldering effect, inverted *T* wave. Diagnosis: interatrial defect, *cor pulmonale*.

regarded as indicative of Lutembacher's syndrome of associated mitral stenosis. The general opinion is that the bruit is produced by increased flow in the pulmonary artery, and in agreement with this is systolic in time. However, one would then expect the bruit to be always maximal in the second left intercostal space. This is not the case. It is not inconceivable that movement of blood

between the atria may play a part in the production of the bruit, since much blood does pass between the atria, and systole with its vigorous downward movement of the atria may aid this. I consider the same "rrupp" quality is heard in the typical mitral presystolic bruit at the mitral

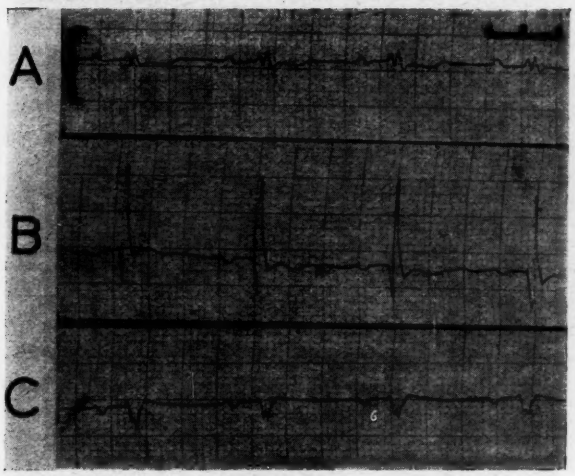

FIGURE V.

The electrocardiograms of leads A, B and C in patient P5. Note very low voltage in leads A and C (the patient had severe myocardial disease) with flat and biphasic *T* wave. Note the extra *R* wave in lead A, and the large *Q* wave in lead B due to right cardiac strain.

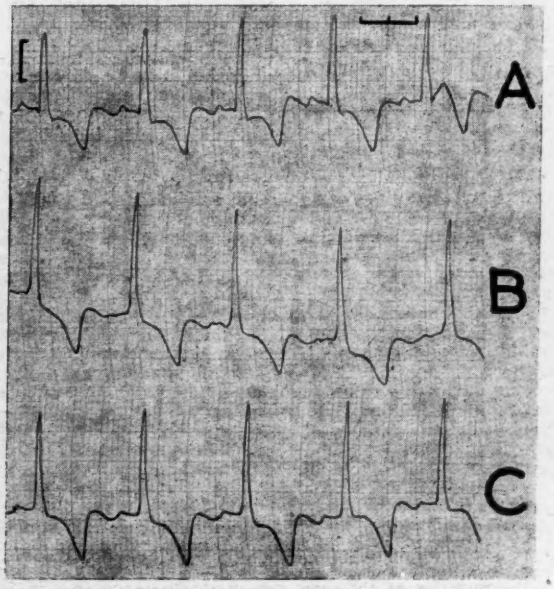

FIGURE VI

The electrocardiogram of leads A, B and C of patient P9 with pure pulmonary stenosis. Note the large *R* waves with no extra wave formation in lead A, the absent *S* wave and inverted *T* wave. This is a classical tracing of pulmonary stenosis.

area. This is also a *crescendo* bruit, and comes from the depth of the heart and not just under the rib layer. It may be that these features account for the similarity of the bruit quality to that of interatrial defect. On the other hand, the systolic bruit of pulmonary stenosis sounds more superficial and in my experience has a different "quality".

Figure VII illustrates the phonogram of patient P1, a female, aged fifty-six years, who was regarded as having a systolic third left intercostal murmur associated with interatrial defect. A late presystolic element could be heard, and this is shown in the phonocardiogram at the arrow,

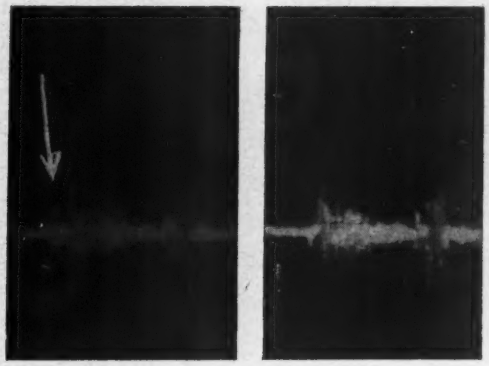

FIGURE VII.

Phonocardiogram of patient P1. Note the *diminuendo* systolic bruit immediately following the loud first sound heard in the third left intercostal space next to the sternum. At the arrow a very late presystolic bruit was noted in one cycle.

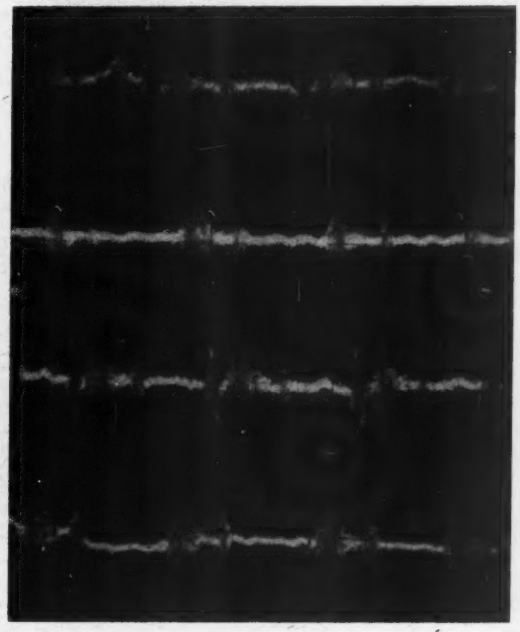

FIGURE VIII.

Phonocardiogram of patient P2 with interatrial defect. Top panel: electrocardiogram lead I with phonocardiogram from third left intercostal space next to the sternum. Second panel: phonocardiogram alone. Third panel: electrocardiogram lead I with phonocardiogram in third left intercostal space further to the left. Bottom panel: phonocardiogram alone. Note the rapid *crescendo* with less rapid *diminuendo* systolic bruit.

though not in all cycles (Figure VII). There was no other evidence in this patient that mitral disease was present. There was no mitral presystolic bruit, and the heart therefore should probably not be regarded as being of the Lutembacher type. More importantly, the bruit has a "slappy" sound, with rapid *crescendo* and slower

diminuendo (Figure VIII). Fluoroscopic examination revealed increased pulmonary vascular pulsation, and an X-ray examination (September 17, 1951) revealed pronounced enlargement of the heart to the left and right. Prominence was noted in the region of the pulmonary artery, and there was a normal aortic shadow, with considerable congestion throughout both lungs. Scoliosis of the dorsal vertebræ and normal ribs were present.

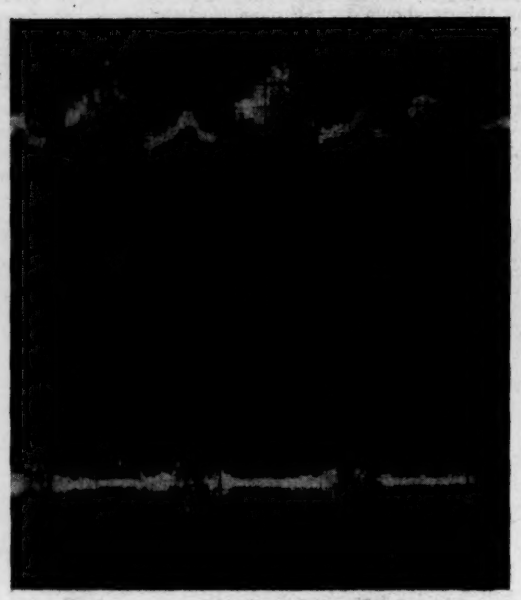

FIGURE IX.
Phonocardiogram of patient with pulmonary stenosis. Upper panel: electrocardiogram (lead I) with phonocardiogram. Lower panel: phonocardiogram alone. Note the long late systolic bruit of fairly well maintained amplitude.

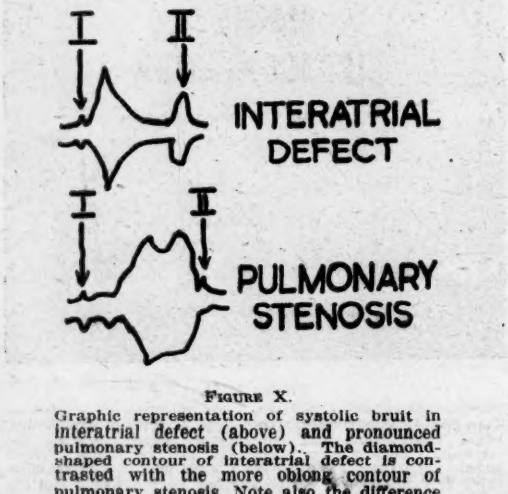

FIGURE X.
Graphic representation of systolic bruit in interatrial defect (above) and pronounced pulmonary stenosis (below). The diamond-shaped contour of interatrial defect is contrasted with the more oblong contour of pulmonary stenosis. Note also the difference in timing: I and II, first and second heart sounds.

In another patient, P2, who had a pronounced systolic bruit in the third left intercostal space (and also a diastolic murmur conducted down the margin of the sternum), the systolic bruit again had the typical quality of an interatrial defect murmur, and once more the phonocardiogram shows a rapidly rising *crescendo* with more gradual (but still quite rapid) *diminuendo*. The general outline is diamond shaped, as shown graphically in Figure X. Further details concerning the establishment of the diagnosis in this case appeared above. The point to be made here is the difference in quality, timing and "shape" of the bruit compared with that of pronounced pulmonary stenosis (Figure IX). (The distinction from mild pulmonary stenosis may not be so clear-cut.) This phonocardiogram was obtained from P9, a female, aged twenty-three years, who had never been able to play sport as vigorously as her fellows. A very harsh systolic bruit was heard, maximal in the second left intercostal space to the left of the sternum. Fluoroscopic examination revealed considerable prominence of the pulmonary artery with moderate pulsation. X-ray examination showed the heart to be at the upper limit in regard to size, with a very prominent

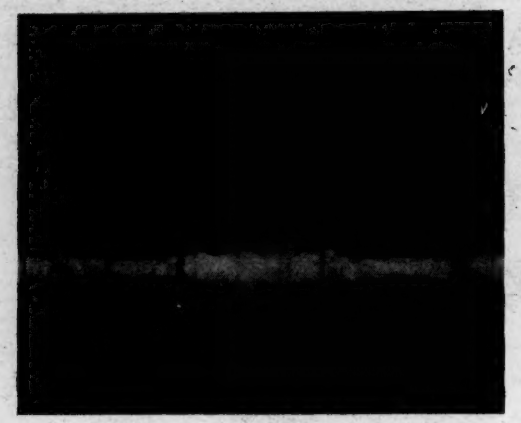

FIGURE XI.
Phonocardiogram of patient P10, pulmonary stenosis. Note the rather oblong-shaped systolic bruit.

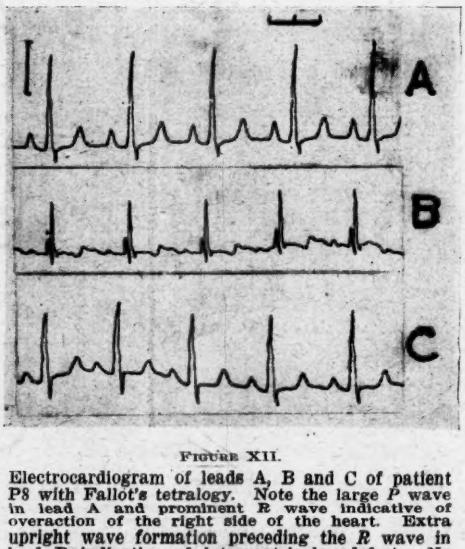

FIGURE XII.
Electrocardiogram of leads A, B and C of patient P8 with Fallot's tetralogy. Note the large *P* wave in lead A and prominent *R* wave indicative of overaction of the right side of the heart. Extra upright wave formation preceding the *R* wave in lead B indicative of interventricular defect. No step on the *R* wave in lead A—that is, no interatrial defect.

pulmonary artery shadow. The blunt and rather high apex of the heart suggested right ventricular enlargement. There was no pulmonary plethora. The aortic shadow was small and the appearance was consistent with the diagnosis of pulmonary stenosis. The phonocardiogram (Figure IX) showed the bruit to be late in systole extending right up to the second heart sound; this has previously also been described by Evans (1951) and Leatham (1952) in severe pulmonary stenosis, extending beyond the aortic second sound. The additional point to be made here (apart from the timing) is that the contour of the bruit represented graphically is more oblong in shape than that of interatrial defect (Figure X). Even in pulmonary stenosis when the bruit occurs earlier (as it may) the oblong contour may still be apparent, as is shown in the bruit of P10, a female, aged eighteen years (Figure XI); the second sound is unusually delayed in this instance.

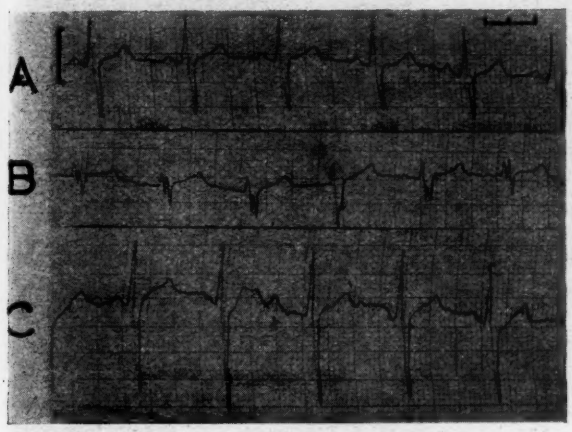

FIGURE XIII.

Electrocardiogram of leads A, B and C of patient P6. Note the extra wave formation in lead B, like an upright *Q* wave followed by a small *R* wave indicative of interventricular defect. This was proven at autopsy.

The phonocardiograph is, I believe, useful in establishing on a firm basis the distinction between these two bruit qualities which I think the ear can detect with the stethoscope; but it is to be remembered that the phonocardiograph does not show the findings as actually heard by the ear through the stethoscope, which amplifies the higher frequencies out of all proportion to the lower frequencies. Further, the phonocardiograph itself, unless made to amplify "logarithmically", does not give an accurate indication of what the ear might hear even if the stethoscope was a wide range (faithful) conductor. In any case, the phonocardiograph may be valuable in training, as was the polygraph originally in distinguishing auricular fibrillation from extrasystoles, and then, after fuller clinical appreciation, the polygraph was not required to distinguish them (Mackenzie, 1914).

Electrocardiographic Findings in Interventricular Defect.

While lead A, which is taken from the *manubrium sterni* and the xiphisternum—that is, from top to bottom of the right and left auricle—shows a typical appearance in the tracing from the patient with interatrial defect, we find that the tracing from lead B, which is taken from the left mid-axilla and xiphisternum, may show a change in interventricular defect. Lead B is aligned over both right and left ventricles. A less evident change has been described in "right" Wilson leads (for example, RV_4) in interventricular defect. This finding in lead B has already been referred to (Trethewie, 1953); the patient was operated on for Fallot's tetralogy (Blalock operation). In this paper the wave was described as an upright Q wave, owing to its timing and to the fact that the *Q* wave is associated normally with septal phenomena in the electrocardiogram. The tracing from this patient is reproduced in Figure XII. Two further cases are recorded here.

Patient P6, a male, aged eighteen years, was regarded clinically as having pulmonary stenosis. On August 20, 1951, an electrocardiogram showed a low voltage *R* wave (0·3 millivolt) in lead B; this showed double wave formation and pronounced notching phasic with respiration (presumably from axis change), which I considered due to a congenital defect. There was also a very tiny step (almost negligible) of 0·1 millivolt on the *R* wave in lead A. Fourteen months later (Figure XIII) the voltage of the tracing had increased slightly, the extra wave form in lead B remained at 0·3 millivolt (which I considered was definite evidence of interventricular defect), and the phasic change with respiration became more pronounced. In view of the timing of this wave occurring 0·10 second after the tip of the *P* wave, while the *R* wave in leads A and C occurred 0·12 second after the tip of the *P* wave, I regard this extra wave form as an upright *Q* wave rather than an *RSR* wave formation.

After operation for pulmonary stenosis this patient unfortunately died. At autopsy he was found to have a huge interventricular defect.

Patient P7, a male, aged fourteen years, was said to have had a heart anomaly since birth. A systolic bruit was heard and described as maximal over the third left intercostal space to the left of the sternum. However, on spatial criteria he appeared to have two bruits: a harsh systolic bruit maximal over the second left intercostal space just to the left of the sternum, and another harsh systolic bruit maximal over the fourth left intercostal space just to the left of the sternum. X-ray examination revealed good diaphragmatic excursion, and no abnormality in the posterior mediastinum; when a barium bolus was given, the findings were normal. There was regular cardiac pulsation of normal depth. X-ray examination showed the lung fields to be clear. There was no dilatation of vessels of the lung or hilum. The heart shadow was within the upper normal limit of size. The apex was blunt and raised above the diaphragm. The aortic shadow was normal. There was some prominence of the right auricle and possibly of the right ventricle, and high on the left cardiac border there was doubtful prominence of the conus of the right ventricle. The electrocardiogram showed in lead A (Figure XIV) a prominent *R* wave; this wave was also prominent in leads B and C. The *T* waves were small and the *S* waves negligible. This is a classical case of right ventricular preponderance (Trethewie, 1953) without the excessive strain of *cor pulmonale* (owing to the absence of a pronounced *Q* wave), and the absence of extra *R* wave formation in lead A indicates that there is no interatrial defect. On the other hand, there is an extra wave formation in lead B, an upright *Q* wave of 0·25 millivolt appearing. This finding is in accord with the finding of a second systolic bruit maximal in the fourth left intercostal space close to the sternum, due to interventricular defect.

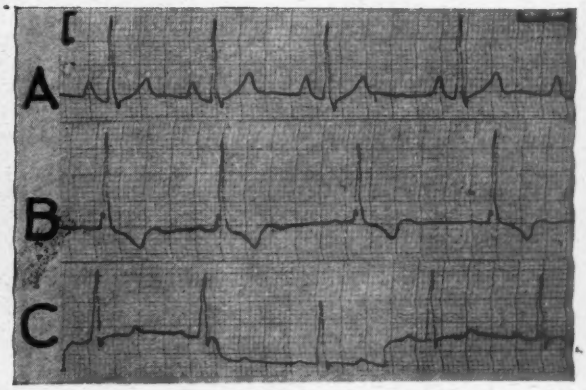

FIGURE XIV.

Electrocardiogram of leads A, B and C of patient P7. Note the large *P* wave in lead A, prominent *R* waves and virtually absent S waves indicative of preponderance of the right side of the heart in a patient with pulmonary stenosis. Note the extra wave formation in lead B at the commencement of the *R* wave (0·25 millivolt) indicative of interventricular defect.

The hæmoglobin value was then estimated and found to be 120% (14·8 grammes *per centum* standard), a rather high figure for an Australian boy of fourteen years. At operation, infundibular pulmonary stenosis (as expected) was detected, but no comment was possible as to whether an interventricular defect was present too, though it was admitted this could not be excluded.

As yet, it cannot be indicated with what frequency the extra wave formation (upright *Q*) occurs in interventricular defect, but as more cases are investigated this will be reported on.

Discussion.

It would appear from the foregoing that the finding of a step wave on the *R* wave of the electrocardiogram (lead A) is a distinctive finding in interatrial defect. In all probability the finding of "partial bundle block", so called, in the standard electrocardiogram in 95% of cases of interatrial defect has a similar origin to the step wave in lead A. The more clearly indicated finding in lead A of the A, B, C display probably is due to the fact that lead A is taken from a point immediately above and a point immediately below the right auricle—that is, immediately over the region of the pathological involvement. The "partial bundle block" appearance in the standard electrocardiogram has not been regarded as pathognomonic, perhaps owing to the variability in the appearance of the contour. Certainly it has not the distinctive high voltage found in lead A; for example, in patient P4 the standard leads indicated "the pattern of right ventricular hypertrophy and pulmonary hypertension". It was thought this was consistent with a diagnosis of pulmonary stenosis. However, owing to the quality of the bruit and the clear-cut findings in lead A of the A, B, C display, I regarded the patient as having definite evidence of interatrial defect. This was later confirmed by catheterization. When in the case of the A, B, C display, the step wave on the *R* wave is coupled with the distinctive quality of the bruit heard stethoscopically and described above, the diagnosis of interatrial defect would appear to be practically certain. In addition, there may be the well recognized findings—for example, the commonly associated wide split in the second sound at the base, pulmonary plethora, hilar dance, prominent pulmonary artery, and information from cardiac catheterization concerning blood oxygen content and maybe movement of the catheter to the left auricle from the right. However, it would appear that cardiac catheterization, which is not without risk, does not usually need to be carried out in such cases to establish the diagnosis. Further work should establish whether the finding of the characteristic step-wave alone is diagnostic.

In view of the proposed surgical interference with repair of the defect in this condition, the accurate diagnosis of a previously untreatable congenital anomaly becomes important, and then the distinctive findings in the A, B, C display are valuable, especially when it is realized that other evidence (Trethewie, 1951, 1953) indicates the value of the A, B, C display alone in other cardiac conditions.

Summary.

Details of the electrocardiographic findings from the use of leads A, B, C in five cases of interatrial defect are presented.

The characteristic features of the tracings are indicated and compared with the tracings in pulmonary stenosis and interventricular defect.

Features of the stethoscopic appreciation of the bruit are discussed in conjunction with phonocardiograms of the bruit in interatrial defect.

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PSYCHOLOGICAL EFFECTS OF PHYSICAL DISABILITY.[1]

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THE psychological effects of physical disability constitute such a large subject that we can hope to deal with only a few of the more important aspects in a short paper such as this.

Important as are these effects when they occur in the adult, they may be of much more serious consequence when they occur in childhood. This is not always realized, as it is often thought that the adult with his responsibilities is much more likely to be adversely affected psychologically than the child, who is dependent and has no obligations to anyone else. Take for instance the case of the labourer, married, with children, paying off his home, who is suddenly incapacitated by a serious accident or by an attack of poliomyelitis—how do the psychological problems here compare with the case of the three or four year old child who suffers a similar illness? In the case of the labourer complex readjustments are necessary—a new way of life, acquiring new means of earning a living, loss of much of his independence, and so on—all putting considerable strain on his mental adaptability. But he has this advantage over the child, and that a not inconsiderable one—namely, his mental maturity. His character and personality are well formed, and therefore he is equipped to deal with the inevitable frustrations and necessity for adaptations. In the case of the child his mind is still in the making—he is a long way from maturity; and what makes the position more precarious for him is the fact that motor activity is a very important means by which mental development takes place.

In our attempt to study the psychological effects of crippling disorders in children, let us first mention some of the salient points in normal mental development.

The instinctual forces that play the most important part are the erotic and the aggressive instincts. There are of course other drives; but the most important from the point of view of mental development are found to be modifications, sublimations or combinations of these two fundamental instincts.

The erotic instincts develop from an autoerotic state through a phase of attachment to the parents and others in the child's early environment, and finally, in maturity, are directed to persons outside the family circle.

The aggressive instincts are first of all destructive, but later are turned towards mastering the environment, seeking new objects, finding substitute gratifications, and generally in maturity are used more in the service of construction than of destruction.

Both erotic and aggressive impulses in children are often associated with much guilt and anxiety. This is due partly to various inner psychological causes, but also partly to our particular culture, which is somewhat intolerant of childhood sexual activity and of aggression, especially if directed against the parents or people in authority.

Another important fact that has a bearing on our subject is the child's different appreciation of reality compared with that of the adult. The mature person sees the world as a place where some things happen according to human design, but where many things happen by chance or according to general laws of Nature divorced from any notion of benevolent or malign purpose. Not so the child. It has to learn this by a slow process of mental growth and education. At first, even when showing considerable mastery of the language so that it may even give the impression of being a little adult, the child is very close to an emotional type of thinking which is characterized by

[1] Read at a meeting of the Victorian Fellows and Members of The Royal Australasian College of Physicians at the Receiving House, Royal Park, on October 18, 1952.

the idea that whatever happens to the child is deliberately caused by some kindly or hostile agency. We see a persistence of this type of mentality in many animistic cultures, in which most events are ascribed to good or bad magic. During the course of development of the sense of reality in the child's mind, this primitive way of thinking and feeling becomes relegated to the unconscious part of the mind. The fact that it may still remain active can be seen in certain adult attitudes which will be mentioned later, and also in certain phenomena in such procedures as deep hypnosis or psychoanalysis during which these primitive stages of mind can be reexperienced.

In the light of these facts, let us now try to appreciate something of what goes on in the child's mind when it suffers some crippling disease such as poliomyelitis. The wish to move about and the fact of being unable to do so create considerable anxiety and puzzlement. Physicians and parents often find it difficult to know just how to explain it to a child. They naturally do not want to increase the child's anxiety by any reference to the most severe consequences, and at the same time they do not want to underrate the condition. References to the condition as, for instance, "a cold" only increase anxiety, as the child soon senses that it is a strange cold indeed that requires speedy removal to hospital, masked nurses and visitors, splints, iron lungs and so on. The separation from the mother, which is inevitable under our present system of hospitalization, is an added anxiety. It is at a time such as this that the mother's presence is needed more than usual. Loving care from the mother is the best antidote yet discovered for anxiety in the child.

In accordance with that primitive tendency to believe that any misfortune is deliberately intended, the child thinks that its illness is a punishment for some misdeed. According to the child's age and development the punisher is regarded as the parents, or God, or some vague notion such as fate. The misdeed may be some secret and forbidden autoerotic activity or some act or thought of aggression or spite against the parents or a sibling.

Now these two kinds of impulse press for expression irrespective of reason or social condemnation, and therefore cannot be ignored. We see that the already present sense of guilt and anxiety tends to be augmented by the onset of a paralytic condition. It is as if the patient says to himself: "If these feelings I have are going to be punished like this I had better try and stop them altogether." Then the impulses tend to become repressed, so that libidinal development is interfered with and the patient becomes unduly timid, showing a distinct fear of being aggressive towards others and even towards activities that do not involve people.

I do not mean to imply that this chain of events always happens. It is obvious that it does not. I mean only that the chances that this course of events will take place are considerably greater in the paralysed child than in others—especially if the psychological handling is not careful.

The tendency to repression of aggression with its consequence of inhibition of activity generally is of particular importance. The possible effects on the patient's character and personality and on the general conduct of his future life are fairly obvious. However, one aspect is not so obvious, and that is that there is a distinct possibility that the sense of guilt about being aggressive may militate, to some extent, against maximal recovery of paralysed muscles. I understand that, according to some authorities, mental alienation is an important factor to combat in the treatment of poliomyelitis. The sense of guilt may contribute materially to this alienation. Be that as it may, I have encountered cases in which I was satisfied that residual weakness was not entirely due to neuronal deficiency, and these were cases in which undue submissiveness was a pronounced feature.

This brings us to another phase of our discussion—namely, the psychological aspects of treatment—and what is said is intended to refer mainly to the treatment of poliomyelitis.

There seem to be two principal schools of thought here: one emphasizes the importance of splinting by day and by night, sometimes for long periods, to prevent deformity and preserve the function of weakened muscles; the other claims to achieve satisfactory results with a minimum of splinting, and, as far as I can see, by encouraging more activity—or, to put it another way, by showing less concern about the dangers of fatigue and over-activity. I am not in any position to have a worthwhile opinion on the relative merits of these two approaches; but we should try to assess the various psychological factors concerned and take them into consideration in any régime of treatment.

There is no doubt that the prevention of deformity is an important object of treatment from the purely psychological point of view. A feeling of aversion for deformities is very widespread. Psychoanalysis can trace this aversion in neurotic patients quite unmistakably to unconscious fear of the deformed person. He is regarded as having been punished for some transgression, and therefore is a stark example to another person of what might happen if he should give way to forbidden impulses. Again, the deformed person is feared because it is thought that he is seeking revenge and is therefore dangerous and has evil intent. These fantasies appear quite ridiculous to a person in contact with reality, and therefore do not appear in conscious thinking. However, their presence gives rise to the feeling of aversion which does appear in consciousness. The link between deformed and evil can be seen quite readily in novels and on the screen—the criminal appears very much more sinister, evil and frightening if he is given a hump back or a club foot. Therefore the person with a deformity has increased difficulty in making an adequate social adjustment owing to his having to overcome or compensate for a feeling of aversion in others. This feeling is often pronounced in neurotic patients, but is also present to some extent in many normal people. The person's reaction to his own deformity varies from feelings of inferiority, shyness and sometimes a gross exaggeration of the supposed effects on others, to feelings of resentment and hatred of normal people.

The beneficial psychological aspects of splinting are fairly straightforward; it is encouraging for the child as part of an interested, kindly and persistent attempt to preserve the good shape of his limbs and make him strong and healthy again.

However, there are psychological difficulties involved, particularly in young children, in the limitation of spontaneous movement, in the enforced inactivity which increases a sense of frustration. Various emotional difficulties may thus be provoked. There is the danger of increasing anxiety and guilt about moving and being aggressive. The feeling of being queer-looking is sometimes increased by splints—for example, having to wear aeroplane splints to school.

The necessity for avoiding over-exertion or fatigue for long periods of time has several disadvantages. Firstly, it is badly tolerated by the very young; secondly, violent exertion plays an important part in a child's mental development. It is his way of relieving pent-up aggression which might otherwise have to be repressed. Aggression that is not externalized is often turned against the self. The possibility of strong physical action enables a person to turn his aggression from forbidden objects to objects not forbidden, with a consequent relief of tension.

If there is anything in the ideas set forth here, then the treatment of poliomyelitis seems to contain some elements of a dilemma. It would seem that certain measures necessary for the avoidance of deformity and the preservation of good muscle function are hard to reconcile with the optimal conditions for mental development. I do not pretend to have an answer.

In this short paper I have tried to set out some of the more important psychological problems involved, but have said nothing about what we should do about them. At least I hope that to state the problems is some way along the road to solving them.

This is a subject that still requires much research. I think it calls for much patient collaboration between the various disciplines involved before we can be certain that we are doing the best of which we are capable in the task of rehabilitating those who have suffered a severe crippling disorder.

Reports of Cases.

HYDATID CYST OF THE ORBIT: REPORT OF A CASE.

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HAMILTON (1952), in a review of a series of orbital tumours occurring in Australia, drew attention to the only recorded case of hydatid disease of the orbit reported in this country (Pockley, 1916). An additional case is now described.

Clinical Record.

A male patient, aged fifty-five years, was first examined as a private patient on August 26, 1949, when his sole complaint was headache. In the course of routine ocular and refractive examination he was found to have one prism dioptre of hypophoria in the left eye. No orbital tumour was detected. He again presented on December 22, 1949, when he was found to have two prism dioptres of hypophoria in the left eye, and on firm palpation a tense swelling was obvious in the upper nasal quadrant of the left orbit. The bony margins of the orbit were felt to be intact. Examinations by a rhinologist and X-ray examination gave essentially negative results. The Wassermann test also produced a negative result, but the response to the Casoni skin test was positive. There was no evidence of hydatid disease elsewhere. A provisional diagnosis of orbital hydatid cyst was made.

Operation was carried out at the Victorian Eye and Ear Hospital on January 25, 1950, through a skin incision above the medial angle of the eye. The unilocular cyst was located outside the muscle cone. The fibrous adventitious coat was adherent to neighbouring soft tissues, but it was possible to remove its lining sac. Convalescence was uneventful.

The patient was last examined on February 1, 1955, when there was no hypophoria and no evidence of hydatid disease. With correcting lenses the visual acuity of each eye was 6/6.

Microscopic Examination.

The tissue submitted for pathological examination was laminated membrane with attached areas of granulation tissue, fibrous tissue, cholesterol crystals, giant cells and hæmorrhage. The diagnosis was hydatid disease.

Discussion.

Holland (1948), writing of hydatid disease of the orbit, states that "text-books have very little to say regarding this condition". His experience in India was of eight cases, and, as in the one now reported, the cysts were "generally on the nasal side and always in the upper fornix". He comments on the absence of a pericystic membrane. However, the case here reported resembles the three reported from Egypt by Handousa (1951) in that a thick adventitia was present. Handousa's three patients were aged between nine and twelve years.

Summary.

The clinical and pathological findings are reported of the second case of hydatid disease of the orbit recorded in Australia. The rarity of this condition in a country where hydatid disease is far from infrequent may be regarded as justification for the publication of this case.

Acknowledgement.

My thanks are due to Dr. H. Ryan for the Casoni test and the pathological report of the cyst.

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VOLVULI OF THE SIGMOID COLON COMPLICATING PREGNANCY AND CAUSING STRANGULATION OF THE UTERUS.

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IN view of the extraordinary findings in this case, it was thought that it should be recorded in the medical literature.

Clinical Record.

Mrs. A., aged thirty-three years, with one child, aged sixteen months, was admitted to The Women's Hospital, Melbourne, at 9.30 a.m. on July 3, 1953. She had had no abortions or abdominal operations. Her last menstrual period had occurred on January 28, 1953, and her estimated date of confinement was therefore November 4. The course of her pregnancy until her admission to hospital had been uneventful except for three attacks of vomiting and diarrhœa in the more recent weeks. These had been diagnosed as gastro-enteritis. The last attack of vomiting and diarrhœa had ceased on July 2. At 6.45 a.m. on July 3 she awoke with a dull backache. She got up and passed urine, but did not pass flatus or fæces. She went back to bed, and abruptly the backache became very acute. This was followed by severe lower central abdominal pain. She had not vomited up to the time of arrival at the hospital.

On examination, the patient was semi-comatose and complaining of low backache. She was pale and in a state of extreme shock and collapse. She was pulseless, no temperature was registered, the systolic blood pressure was 60 millimetres of mercury and the diastolic blood pressure could not be recorded. The abdomen was found to be grossly distended. The entire left side of the abdomen was felt to contain dilated bowel; it was soft and tympanitic, there were no bowel sounds, and no tenderness was noted. The uterus was felt pushed out to the right flank, and in size corresponded to a twenty-six weeks' pregnancy—that is, it was larger than could be accounted for by the period of amenorrhœa. It was not tender. No fœtal parts were felt and the fœtal heart was not heard. Rectal examination revealed a grossly dilated rectum, and no mass was felt outside it. The passage of a flatus tube failed to produce any escape of gas. Vaginal examination revealed that the cervix was pulled up almost out of the pelvis. On later examination, the cervix could not be felt. Catheterization of the bladder produced less than one ounce of normal urine.

Half an hour later it was noticed that the uterus had increased in size, and then it enlarged, rapidly and visibly, until the fundus was under the right costal margin. The distension on the left side of the abdomen had not increased.

A blood transfusion was commenced and the patient's general condition rapidly improved. By midday, the blood pressure was 100 millimetres of mercury, systolic, and 70 millimetres, diastolic. The pulse rate was 70 per minute; the pulse was regular and of good volume.

A provisional diagnosis of acute bowel obstruction, probably volvulus, with an associated concealed accidental hæmorrhage in the uterus was made; but correlation of the double diagnosis was difficult, and many suggestions were made for a single diagnosis, the following being considered: concealed accidental hæmorrhage with superadded ileus, torsion of the uterus with consequent accidental hæmorrhage and ileus, and torsion of the uterus with a volvulus.

Although the patient's general condition had not deteriorated, the early improvement having been maintained, it was decided to perform laparotomy, and at 7.30 p.m. on July 3 the abdomen was opened. The findings were as follows. The entire left side of the abdominal cavity was found to be occupied by an enormously dilated segment of sigmoid colon, about two feet in length by one foot in width; this bowel was purple, with widespread subserous ecchymoses involving both bowel and mesocolon. The *appendices epiploicæ* were œdematous and black. An abnormally long colon had become twisted around the uterus and upon itself in the following fashion (see Figure I). The lowest foot or more had doubled; the apex of the

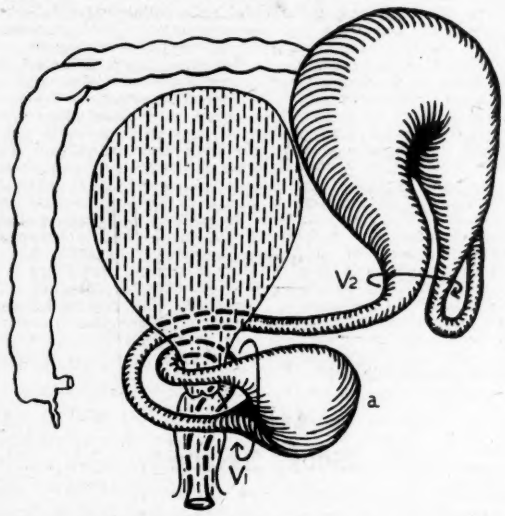

FIGURE I.

loop (a) thus formed had passed from the left side of the pelvis behind the uterus, over the right broad ligament, in front of the uterus, to the left side of the pelvis again, forming a volvulus of the colon (V1) there, and thus constricting the base of the uterus. Above this the remainder of the abnormally long pelvic colon had formed a volvulus (V2) by twisting around itself twice in a clockwise direction. Thus there were two separate volvuli of the sigmoid colon, the lower one causing strangulation of the uterus. The uterus itself was deviated towards and occupied the entire right side of the abdominal cavity. It was enlarged to the size of a thirty weeks' pregnancy and was pulled upwards out of the pelvis by the taut constricting loop of bowel. The uterus was purple in colour. The broad ligaments, ovaries and Fallopian tubes were greatly congested.

The distended colon was deflated by a stab incision made with a trocar and cannula, and the constricting loops of bowel were released. The uterus and adnexæ immediately showed signs of blood flow by becoming pink and decreasing in size. The affected portion of the bowel was considered viable and replaced.

The uterus was emptied by hysterotomy. This was done to prevent pressure by it on the pelvic colon as it returned to the pelvis, and also to avoid the strain of labour on both the repaired abdominal wall and the systemic circulation, as the patient was now beginning to show evidence of shock. A dead fœtus of about twenty-four weeks' development was delivered. The placenta was lying free, and there were 30 ounces of blood and blood clot in the uterine cavity.

On leaving the operating table, the patient was pale and clammy; her pulse rate was 120 per minute, and her blood pressure was 90 millimetres of mercury, systolic, and nil, diastolic. Blood transfusion was continued during the operation and throughout the night.

During the first critical twenty-four hours, the patient showed some improvement. However, despite blood transfusion, intravenous serum and electrolyte infusions, chemotherapy and duodenal suction, this improvement was not maintained. The temperature remained elevated to about 102° F., the pulse rate was about 130 per minute, and the respirations numbered 30 per minute. The blood pressure was steady at 130 millimetres of mercury, systolic, and 70 millimetres, diastolic, and the hæmoglobin value was maintained at about 90% (13 grammes per 100 millilitres). The abdominal distension became gross, fæcal fluid was aspirated via the duodenal suction tube, and blood was passed *per rectum*.

On July 20, seventeen days after the patient's admission to hospital, it was decided that further surgical intervention was necessary, and a large intraperitoneal collection of pus was drained through an incision in the left iliac fossa. At the same time a stab sigmoid colostomy was established, and rubber drain tubes were left in the colon and the peritoneal cavity.

Improvement in the patient's condition was evident within twelve hours. This improvement continued, and at the end of one week after the second operation, intravenous therapy and duodenal suction were stopped. Meanwhile all the tubes had been draining freely, and these were allowed to fall out of their own accord at the end of the second week. The colostomy continued to act freely.

Then began a period of slow convalescence. A small bowel motion occurred *per anum* on August 18, and, although the colostomy continued to drain, normal bowel motions became a daily occurrence. On September 8 the patient was allowed to go home.

The patient was examined regularly in the out-patient department. On November 6 it was noticed that the colostomy had actually closed, although a sinus remained at the colostomy site from which there was a small amount of mucopurulent discharge. Normal bowel actions were occurring once or twice daily. There was no toxæmia, and the patient had regained her usual weight and was eating a normal diet.

Summary.

1. A case is presented of mechanical strangulation of a pregnant uterus caused by the sigmoid colon, with associated volvuli of the latter.

2. It is presumed that an abnormally long and mobile sigmoid colon slipped over the uterus as it was rising out of the pelvis at about three months' gestation.

3. The vomiting and diarrhœa occurring in the weeks preceding the actual obstruction were probably pre-obstructive symptoms, and due to pressure on, and irritation of, the large bowel.

4. When the volvuli did actually form, the distension in the obstructed loops of bowel became so great as to cause the uterus to be lifted high up into the abdominal cavity by mechanical traction of the bowel around the base of the uterus. At the same time, this strangulated segment of bowel was drawn so taut that it obstructed the blood supply of, and particularly the venous return from, the uterus, thus initiating the concealed accidental hæmorrhage which resulted in the death of the fœtus.

Acknowledgements.

I wish to thank Professor Lance Townsend for permission to publish this case, and Mrs. C. Aldor for preparing the diagram.

INFANTILE ECZEMA IN A BREAST-FED BABY: RELIEF FROM GOAT'S MILK.

By R. W. NICHOLLS,
Yackandandah, Victoria.

BABY A was born on March 10, 1954, and fed on breast milk only till October 1. On March 21 he underwent a Ramstedt operation for pyloric stenosis, but recovered well and increased his weight rapidly. On about May 10—that is, at the age of two months—he developed the typical red weeping pruritic rash of infantile eczema on the scalp and limbs. The rash rapidly extended and became practically universal in distribution.

Hydrocortisone ointment 1% (Ciba) was applied to the left side of the scalp and the left upper limb, and proved effective in reducing the redness, weeping and itching. The contrast with the untreated right side of the scalp and right upper limb was plain to see. Despite improvement with the hydrocortisone ointment, the eczema remained severe whilst the baby was fed on breast milk only.

On September 29, after an intradermal test, a dose of 10 international units of corticotrophin (Commonwealth Serum Laboratories) was injected intramuscularly. This was followed in a few hours by the appearance of large erythematous patches and many urticarial weals, but with temporary improvement in some of the weeping areas on the scalp and limbs.

Feeding with goat's milk for three feeds a day was commenced on October 1. By the next day the baby seemed to have less pruritus and had a distinctly better and happier look about his eyes. Three days after the commencement of goat's milk feeding, it was obvious that the rash was much relieved. He still had two breast feeds per day until November 3, but thereafter continued solely on goat's milk mixture and added vitamins. When he was examined on November 12 his skin was clear except for a patch of scaling on the outer surface of both legs; but the patches were not weeping or angry. In January, 1955, he developed *dermatitis herpetiformis*.

The baby has an aunt who suffers from dermatitis on contact with paspalum grass, and another aunt who is sensitive to kapok.

Dennis G. Brown and Roy L. Holman, of Leeds, report (1953) that no effect on the course of eczema in nine children was obtained by goat's milk feeding.

Reference.

BROWN, D. G., and HOLMAN, R. L. (1953), "A Trial of Goats' Milk in Eczema in Childhood", *Brit. M. J.*, 1: 1202.

Reviews.

Primary Love and Psycho-Analytic Technique. By Michael Balint, M.D., Ph.D., M.Sc., L.R.C.P., L.R.C.S.; 1952. London: The Hogarth Press and the Institute of Psycho-Analysis. 8¾" x 5½", pp. 288. Price: 30s.

THE author is a trained analyst practising in London. His analytical background stems from Ferenczi, and his school, in Hungary. Like Ferenczi, he shows in his work a strong independence and originality of thought. Thus logically enough, he finds himself amongst those analysts who consider themselves independent of the two main schools of thought in London. That is, he accepts much but not all of the newer Kleinian ideas in theory and practice and considers them a development of the more orthodox Freudian ideas rather than a contradiction. This collection of papers contains his criticisms of some of the basic Kleinian notions.

While this book is of importance to practising analysts primarily, there is much in it to interest the general reader and psychologist on the theoretical side. In the first chapter the author traces the development of the erotic instincts and places theory firmly on its biological foundation. This is very salutary these days when so many psychologists, lacking a fundamental training in the biological sciences, play down the importance of the biological foundations of behaviour.

The principal theme of the book is the relationship of love and hate. It is the central problem of the individual as well as being of great importance in the wider field of sociology.

In Chapter 5 there is an interesting discussion on the earliest stage of object love, and its relationship to erotogenic zones, with important implications for the mental health of the infant. It is a subject worthy of the closest study for its bearing on the mental health and stability of adults.

In Chapter 11 Balint deals with the problem of the mechanism of cure in psychoanalysis. He shows that there is much more to it than the recovery of infantile memories, and is well aware that the last word has not been said on this difficult topic.

Handbook of Medical Treatment. Edited by Milton J. Chatton, A.B., M.D., Sheldon Margen, M.A., M.D., and Henry D. Brainerd, A.B., M.D.; Fourth Edition; 1954. Los Altos, California: Lange Medical Publications. 7" x 4", pp. 576. Price: $3.00.

WITH the assistance of several new collaborators the text of this book has been considerably revised in the present edition and its size has been increased. As before, the importance is emphasized of adequate diagnosis before the institution of treatment, and rational rather than empirical treatment is encouraged. The first four chapters deal with general aspects of medical treatment, with fluid and electrolyte therapy and parenteral feeding, with general symptomatic treatment and with dietetics and nutrition. Then follow chapters on the treatment of diseases of the various regions and systems of the body according to orthodox grouping. The concluding chapters are concerned with hormones and hormone-like agents, neoplastic diseases, venereal diseases, infectious diseases, chemotherapeutic agents, diseases of unknown ætiology, diseases due to physical agents, and diseases due to toxins, and there is an appendix on the rehabilitation of the hemiplegic. The descriptions of treatment are in detail. The general arrangement and indexing of the material make for easy reference. The treatment described appears to be orthodox and up to date, and the busy practitioner should find this handbook convenient and useful.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Morals and Medicine: The Moral Problems of: The Patient's Right to Know the Truth, Contraception, Artificial Insemination, Sterilization, Euthanasia", by Joseph Fletcher with a foreword by Karl Menninger, M.D.; 1955. London: Victor Gollancz, Limited. 8" x 5", pp. 250. Price: 20s. 6d.

The author is professor of pastoral theology and Christian ethics at the Episcopal Theological School, Cambridge, Massachusetts, United States of America.

"The Physiological Basis of Medical Practice: A Text in Applied Physiology", by Charles Herbert Best, C.B.E., M.A., M.D., D.Sc. (London), F.R.S., F.R.C.P. (Canada), and Norman Burke Taylor, V.D., M.D., F.R.S. (Canada), F.R.C.S. (Edin.), F.R.C.P. (Canada), M.R.C.S. (England), L.R.C.P. (London); Sixth Edition; 1955. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10" x 7", pp. 1370, with three plates in colour and 80 text figures. Price: £6 9s.

First published in 1937.

"Connective Tissues: Transactions of the Fifth Conference, February 8, 9 and 10, 1954, Princeton, N.J.", edited by Charles Regan, M.D.; 1954. New York: Sponsored by the Josiah Macy Junior Foundation. 9¼" x 6¼", pp. 222, with 55 illustrations, a few in colour. Price: $4.25.

The subjects discussed are: "The Exchange of Materials Between Blood Vessels and Lymph Compartments", "Interstitial Water and Connective Tissues", and "Hormonal Effects on Connective Tissues".

The Medical Journal of Australia

SATURDAY, MAY 28, 1955.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

A SURGICAL JUBILEE.

No surgeon worthy of the name, and indeed, no thoughtful practitioner of medicine in any of its branches, will allow to pass unheeded the golden jubilee which is being celebrated by the well-known and highly respected journal, *Surgery, Gynecology and Obstetrics*. The issue of January, 1955, which is Number 1 of Volume 100, appears suitably attired in a golden coloured cover, and in addition to the usual type of article published in any number, contains reference to the early history of the journal. This journal owes its existence to the imagination and initiative of Franklin H. Martin, who conceived his idea in December, 1904. The early history is told in a signed article by the present editor, Dr. Loyal Davis. This article is preceded by two full-page portraits, one of Franklin Martin and the other of Allen B. Kanavel, the first editor. In the first volume of his autobiography, "The Joy of Living", Franklin Martin describes the birth of *Surgery, Gynecology and Obstetrics*. He writes that in 1904 the cauldron of old-style medical journalism was boiling dry and a new order of service, liberated from commercialism, was demanded. He points out that *The Journal of the American Medical Association*, which was launched by Nathan Smith Davis in 1883, was the one existing medical journal that was divorced from extraneous financing. (Obviously he is referring to America only.) Martin's own experience with articles that he had presented for publication had made him realize that "the medical profession was being exploited by the publishers of scientific periodicals". He was convinced that this unfortunate condition could be remedied if the profession was to organize a practical journal for practical surgeons, edited by active surgeons instead of *littérateurs* only remotely connected with clinical work. Martin invited to his home four young men who had been associated with him in their surgical training—Alan Kanavel, Frederick Besley, William Cubbins and John Hollister. He put his ideas before them and they responded at once with contributions of $500.00 a head which they made as an investment. Of the stock of the Surgical Publishing Company, 49% was distributed among the young associates, and the controlling interest was kept by Martin and his wife. Kanavel, who was named associate editor, was responsible for the editorial policies of the journal, and Martin, known as managing editor, acted in this capacity until he died. A start was made with a subscription list of about 600, and in eighteen months the number of paying subscribers was 2800. Since that time, the journal has gone from strength to strength. In the late twenties, Martin purchased the shares of stock which had been issued to his associates at the beginning of the venture, and added a generous bonus. The journal then became the sole property of him and his wife. When Martin died in 1935, Mrs. Martin alone owned the stock of the journal, and at her death the shares and reserve of the Surgical Publishing Company became by terms of Martin's will the sole property of the American College of Surgeons. The Franklin H. Martin Memorial Foundation was also created to enable the earnings of the journal to be used for the education purposes of the College. The journal at present is administered by a board of directors, elected by the Regents of the College, and an administrative board consisting of the editor, the general manager and the comptroller of the College. It was planned at the outset that the literary scope of the journal would include the making of a wide survey of surgery, gynæcology and obstetrics of the world. Original articles were to be based upon practical, experimental and statistical subjects and would constitute the major portion of the magazine. As we know, each issue of the journal also includes abstracts from American and foreign journals, reviews of books and subjects which are dealt with editorially. Society reports also find a place.

While all who are interested in the development of surgery and in the dissemination of fresh surgical knowledge will join in offering congratulations and the heartiest of good wishes to the body controlling *Surgery, Gynecology and Obstetrics*, this expression of good will must be rather sterile unless it is accompanied by a looking backwards at the history of past achievements in surgery and a looking forward to possible developments in the future. Retrospect and prospect are exercises which should be undertaken from time to time by all who practise medicine, not only in the wider fields of medical and surgical endeavour, but in the restricted fields of their own practices. When THE MEDICAL JOURNAL OF AUSTRALIA celebrated its silver jubilee in 1939, Sir Henry Newland, at the request of the Editor, contributed an article to the special number entitled "The Changed Outlook in Surgery". This article will well repay study at the present time. Sir Henry Newland laid emphasis on the need for experimental investigation in surgery and also on the part which should be played by physiology. He pointed out that Lister was "essentially and fundamentally" a physiologist. He wrote that had Lister not been, in the first place, a

physiologist, it might be doubted whether he would have lighted upon antiseptic surgery. In the special issue of *Surgery, Gynecology and Obstetrics* there appears an article by L. N. O. Whipple, entitled "An Old Surgeon Views the Surgery of Today". Whipple, like Newland, refers to the place of physiology in regard to surgery. He mentions the recognition during World War I of the importance of physiology in the study of shock and hæmorrhage. He points out that Evarts Graham was one of the first American surgeons to abandon surgery after an internship to study chemistry for three years in order to obtain a better understanding of the physiology and chemistry of surgical problems. We know now that a knowledge of chemistry, physiology and bacteriology is indispensable to the mature development of a surgeon. Whipple names the factors which have increased the safety of surgery and widened the extent and radical character of operations in the various systems. His first is anæsthesia, his second is blood transfusion, and his third is the maintenance of protein, fluid and electrolyte balance. Then he names the antibiotics, but here he condemns "the indiscriminate administration of large doses of antibiotics in shotgun fashion, without first determining the susceptibility of the invading organism to one or more of the antibiotics" as both stupid and unintelligent. Then comes the improvement of surgical hospital facilities. He makes reference to the hospital standardization programme of the American College of Surgeons. It is hoped that the time may come when something of this sort will be attempted in Australia. Whipple then names the educational programme of the American College of Surgeons and the recognition of safe, deliberate technique. Translating this to our own affairs, we may refer to the educational programme of our Australasian and British Colleges of Surgeons. It may reasonably be claimed that one of the most important developments in surgical practice during the last few years has been the acknowledgement of the importance of the apprenticeship system and its adoption in this country as a *sine qua non* of the Royal Australasian College of Surgeons. It is really impossible to separate a retrospect from a prospect in the consideration of surgery and its development. This becomes more evident when we consider the apprenticeship system in relation to the more restricted specialist fields in surgery.

J. Garrodd Allen begins his contribution to the special number of *Surgery, Gynecology and Obstetrics*, entitled "A Younger Surgeon Views the Surgery of Tomorrow", with a quotation from Osler: "Throw away, in the first place, all ambition beyond that of doing the day's work well." He points out that it is impossible, and never has been possible, to predict successfully the future of surgical developments for even short periods of time. He holds that the future of surgery is better served "not by seeing what lies dimly in the distance, but by doing what lies clearly at hand". Allen takes us back to the idea suggested by Sir Henry Newland in 1939, for he writes that there is good reason to believe that the future of surgery and the future of physiology will for several reasons run in parallel directions, if indeed they do not actually merge at numerous points. He thinks that in the future surgery will lean more heavily upon physiology. This need not prove awkward, he declares, for it comes at a time when the classical physiologist is leaving the far from completed field of organ physiology in favour of cellular study. If the surgeon is to learn more of organ physiology, he is likely to be forced into the position of being his own teacher and of doing his own research. Allen states that the migration will be for the biochemist to move into biophysics, the physiologist to become tomorrow's biochemist, and the surgeon to assume at least a dual role—that of surgeon and physiologist. Allen goes so far as to declare that it is becoming increasingly clear that little is known of the physiology of man, and that many of our past views on general physiology may need serious revision in the light of the newly acquired facts that the surgeon is able to provide. We do not need to be reminded that surgery is both an art and a science, nor to recall the fact that one who is a human carpenter and not a scientist cannot be styled a surgeon. Surgeons admitted to the fellowship of the Royal Australasian College of Surgeons are reminded of this fact on their admission, when the President expresses the hope that in addition to practising the art, they will help to advance the science of surgery.

One final point must be made, a point which has been true of surgery in the past and which will continue to be true of it in the future. A surgeon needs to be a good technician, and he must have a trained scientific outlook, but he must do more than this—he must be a man of human understanding for his patients and of ethical standing amongst his colleagues. This point was made in a telling fashion by I. S. Ravdin in a presidential address to the Philadelphia Academy of Surgery in January, 1954.[1] Ravdin thinks that those responsible for the admission of young men for training in medical schools and hospitals in the field of surgery should see to it that only those are admitted who can abide by the golden rule. "Those who would do unto others not as they would wish to be done by, or have those who are near or dear to them done by, should be dropped out of our training programs, for they are lacking in that essential quality which is so necessary to all surgeons—honesty and integrity of effort." Ravdin thinks that it is not too difficult, if one lives with his trainees, to ascertain which of them will be great in heart and soul as well as in surgical technique. It is not difficult, he declares, to find out those who have no essential respect for the dignity of their patients. It is not difficult to find out those who, while searching for the truth, realize the faith which the patient has placed in them. None of us can tell the extent of the fields which will yet be entered by surgery, but we need have no fear of the future if it is left in the hands of those of upright character and of high emprise.

Current Comment.

TRAUMA, STRESS AND CORONARY THROMBOSIS.

THE relationship of coronary heart disease to trauma and stress, whether of physical or emotional origin, presents constantly recurring and ever varying problems. It has often been referred to in these columns, but the importance of the subject warrants reference to a paper read by Alan R. Moritz[2] in the session on legal medicine

[1] *Ann. Surg.*, December, 1954.
[2] *J.A.M.A.*, December 4, 1954.

at the American Medical Association's annual meeting last year. No one doubts, of course, that stress and trauma will on occasion damage the heart, especially the heart that is already defective. However, the problem at issue, at least from the medico-legal point of view, is not so much whether it is possible for an injury to cause or contribute to coronary insufficiency or to heart failure. Rather, as Moritz states, it lies in the criteria for evaluating the degree of probability that such a causal relationship exists in any given instance. Moritz points out that trauma may damage the heart by either of two mechanisms. In one, the effects are direct, in the sense that the disruptive forces of the original impact, whether blunt or penetrating, are transmitted directly and immediately to the heart. In the other, the mechanism is indirect, in that the heart is damaged by the systemic disturbances secondary to the trauma, rather than by the original direct effects of mass in motion. Direct injury to the heart that may result from trauma to the thorax is summarized under the headings of commotion, contusion, laceration, valvular avulsion and coronary artery occlusion; these need no comment beyond pointing out that the last of these must be exceedingly rare. Turning to the second mechanism Moritz states that pathologists who have had the experience of performing autopsies and reviewing case histories from the coroner's or medical examiner's office in an urban area are well aware that an episode of stress, whether resulting from effort, pain or emotional excitement, may precipitate a state of acute coronary insufficiency in a person whose coronary arteries are narrowed by atherosclerosis. The work of the heart may be significantly increased as a result of an episode of physical exertion or emotional excitement, and during such an episode there is characteristically an increase in the rate and amplitude of the heart beat as well as in the systolic blood pressure. However, there is no evidence that such an episode ever causes any permanent damage to a normal heart. It may precipitate the failure of a heart in which the coronary arteries are narrowed by disease, or in which the myocardium has been damaged by previous episodes of coronary insufficiency. It is not necessary for either the coronary disease or the myocardial damage to have been previously symptomatic. If the work-load imposed on such a diseased heart is not in excess of its reserve, the resulting cardiac dilatation is physiological and transient. If the work-load is such as to create an oxygen requirement in excess of what the diseased coronary vessels can supply, an attack of *angina pectoris* or of heart failure may be precipitated. At the same time, *angina pectoris* alone does not indicate that the hypoxic myocardium has sustained irreversible injury. It is to be noted that if heart failure or an attack of coronary insufficiency manifested by angina is to be attributed to an episode of exertion or excitement, it should occur during or immediately after the episode in question. With each hour that intervenes between the pressor episode and the development of signs or symptoms of coronary insufficiency, the probability of a cause-and-effect relationship between the two diminishes.

Moritz goes on to state that the finding of coronary thrombosis with or without myocardial infarction at post-mortem examination of a person who has collapsed and died during or immediately after an episode of stress cannot be construed as evidence that either lesion was caused by the events with which death was associated. Microscopic examination of such lesions almost invariably establishes the fact that the thrombus or the infarct, or both, had started to develop hours or days before death occurred, and therefore could not have been caused by the terminal episode of stress. If the event stipulated is clearly unusual and if it was followed by cardiac failure, the relationship may be reasonably clear; but often the event is not sufficiently unusual to distinguish it from other non-occupational stresses that may have occurred about the same time. In this regard, it is noted that lifting a box of 40 pounds' weight from an overhead shelf may not represent any greater exertion than that of sneezing or straining at stool.

Another possible complication of a pressor episode that may result in coronary insufficiency is the occurrence of hæmorrhage into an atheromatous plaque in a coronary artery. These plaques are richly vascularized, the newly formed capillaries in them are often thin-walled and inadequately supported by the lipid debris that surrounds them, and many of the capillaries are in direct communication with the lumen of the diseased artery. Spontaneous rupture of such a capillary, with escape of blood into the plaque in which it is contained, may cause the plaque to swell with resulting narrowing of the lumen and coronary insufficiency. Such subintimal hæmorrhages sometimes occur during an episode of stress, possibly because of the sudden and severe increase in blood pressure elicited by it. Moritz considers that the probability of a cause-and-effect relationship between an episode of stress and coronary insufficiency can be defended if the stress is authentic, and if it is followed almost immediately by coronary insufficiency that was demonstrated at autopsy to have been the result of a recent subintimal hæmorrhage. It is possible that a pressor-induced hæmorrhage of this kind may fail to cause immediate coronary insufficiency, and that many hours or even days may elapse before the subintimal hæmorrhage or the local reaction to it has progressed to the point of occluding the lumen of the artery. However, this type of relationship between stress and coronary insufficiency cannot be established on clinical evidence alone.

Turning to the question of post-traumatic shock, Moritz states that if an injury is severe enough to be followed by shock with hypotension and vascular stasis, the myocardium, like all other tissues of the body, suffers from hypoxia. If the myocardium of such a person is at a disadvantage because of narrowed and rigid coronary arteries, it is especially vulnerable to systemic hypoxia. Moreover, when the blood pressure of a person with coronary disease falls below a certain critical level, coronary insufficiency with thrombotic or non-thrombotic infarction may occur. Thrombosis during shock may take place at sites of lumen narrowing, and is presumably partially contributed to by the already established intimal disease, and partially by the circulatory stasis of shock.

In summary, the position as seen by Moritz is that the heart, whether normal or diseased, may sustain injury by the direct and immediate effects of mechanical violence. Post-mortem evidence tends to show that coronary thrombosis is rarely the direct result of trauma in people who have died a violent death. In the presence of atherosclerosis, various secondary effects of trauma or stress are probably capable either of inducing a state of coronary insufficiency, or of causing the heart to fail by imposing an excessive work load on it.

ATOMIC EXPLOSIONS AND RAIN.

ALTHOUGH ordinarily well-informed persons regard casting the blame on atomic explosions for occasional bad weather as silly, the number of people who, arguing solely on the basis of *post hoc, propter hoc*, do cast such blame is very great, and some of this number apparently carry enough weight (or perhaps they only talk long enough and loudly enough) to make it necessary to produce scientific disproof of their claims. It then becomes apparent that it is necessary to be rather more than ordinarily well-informed to do this, and the task devolves on someone with special knowledge, like Sir Graham Sutton,[1] Director of the British Meteorological Office, who, in a paper on thermonuclear explosions and the weather, makes a detailed scientific approach to the matter. Even he cannot give a final refutation, because there are gaps in the data, and he states: "At this stage the most that can be attempted is an examination of the plausibility of what is, for many people, a firmly held belief." Since a number of experimental explosions were set off in the spring of 1954, Sutton considers first the summer of 1954 in the British Isles. It was cool, wet and dull, more so than the average, but well within the maximum in each respect. At the same time, large areas elsewhere in the world experienced

[1] *Nature*, February 19, 1955.

more cloud and rain and were somewhat cooler than would be expected from long-term averages, but analysis of climate trends, as distinct from averages, suggests that cool wet summers will be more frequent for some years to come. More immediately, the weather in the British Isles showed no specific fluctuations after known explosions, but conformed to the pattern of existing barometric depressions at the time.

After that, Sutton becomes technical. The accepted figure for the kinetic energy of an average cyclonic disturbance is about $3{\cdot}5 \times 10^{16}$ gramme calories, while the kinetic energy of the general circulation of the atmosphere is about 7×10^{19} gramme calories; but the energy released by the largest known atomic explosion was of the order of 10^{16} gramme calories. This is equivalent to adding one rather small cyclonic depression to the atmosphere, or to increasing the kinetic energy of the whole atmospheric circulation by slightly more than one ten-thousandth—certainly not nearly enough to produce increased cyclonic activity all over the earth for several months. As for dust particles, there was no abnormal weather following the explosions of Krakatoa, Mount Pelée or Katmai, any one of which produced infinitely more dust than can a thermonuclear explosion. In any case, dust has little to do with rain, though it might produce a little interference with solar radiation. Ions and non-hygroscopic particles are unsuitable as nuclei of condensation except in states of humidity far higher than are achieved in nature. In support of this aspect of Sutton's discussion, we may cite B. J. Hanson,[1] who, in an article entitled "Design and Evaluation of Large-Scale Rain-Making Experiments", explains that the dominant mechanism in the production of rain involves the growth of ice crystals in a supercooled cloud and their aggregation into snowflakes, which melt and form raindrops; in warmer clouds, coalescence between water droplets causes lighter rain. A deficiency of condensation nuclei in clouds can be remedied by "seeding" with dry ice, silver iodide particles, water droplets or large hygroscopic particles. Nothing of this nature develops as a sequel to thermonuclear explosions.

Finally, Sutton demonstrates that, given the presence of water vapour, the part played by the electrostatic charge carried by a droplet in causing that droplet to increase in size is negligible, even with the high charges developed during thunderstorms, which are much higher than any that could be imparted by radioactive material. But in the present state of our knowledge, Sutton, when pinned down to actual scientific statements, can only conclude that "so far the available evidence points to the conclusion that recent thermonuclear trials cannot be held responsible for any worldwide extremes of weather encountered in 1954".

[1] *Nature*, March 12, 1955.

CONTROL OF LIVER HÆMORRHAGE BY SPLIT-THICKNESS SKIN GRAFTING.

THE control of hæmorrhage from the liver presents a major problem after accidental injury, and, as Frank Masters, Nicholas Georgiade, Charles Horton and Kenneth Pickrell[2] have pointed out, hæmorrhage has always been a limiting factor in resective or traumatic surgery of the liver. Despite the development of absorbable hæmostatic agents, extensive surgery of the liver is performed in relatively few patients by very few surgeons. With all the artificial aids and the almost routine use of strangulating mattress sutures, exsanguination can occur even from biopsies and relatively small resections. In cases of trauma, either from a shearing force or from penetrating missiles, the problem of hæmorrhage is even more acute. Masters and his colleagues state that, clinically, it has been noted that the bleeding from a raw granulating surface can be controlled by split-thickness skin covering. Grafts placed on a raw surface whose granulations have just been surgically debrided act as a hæmostatic agent as well as skin coverage. This clinical observation suggested that split skin might be used in a similar manner to control hæmorrhage from the cut liver surface. A series of animal experiments was undertaken to demonstrate whether or not such grafts could effectively control liver hæmorrhage of both traumatic and resective origin, and thus permanently resurface raw liver parenchyma. Fifteen dogs were anæsthetized, and a constant drip of 5% dextrose in water into a vein was begun. Surgical exploration was carried out through a combined thoraco-abdominal approach, and the split-thickness grafts were taken by a "Brown Electro-Dermatome" 15/1000 to 18/1000 inch in thickness from the abdomen of the dog. The first series of experiments comprised resection of one-half of the presenting middle lobe of the liver. Bleeding in every case was profuse, both arterial and venous. The grafts were sutured (raw surface down) to the surrounding capsule, and no other attempt was made at surgical control of the arterial or venous bleeding from the raw surface of the liver, nor were hæmatomata evacuated from beneath the graft. Similarly, a second series of experiments was carried out to simulate war injuries. An abdominal trocar eight millimetres thick was thrust at random through the thickest portion of the liver. Bleeding in each instance was vigorous. Split-thickness skin grafts were rolled into a tube, with the raw surface out, and passed through the defect. The grafts were anchored by silk sutures, one on each of the superior and inferior surfaces of the liver. In the final series of experiments the middle lobe was manually torn in five animals. The jagged laceration involved the entire thickness of the liver lobule, and bleeding (both arterial and venous) was massive. A split-thickness graft was folded and used as a wedge between the opposing raw surfaces of the torn liver. The grafts were fixed by two silk sutures.

In this series of 15 experiments, there was no operative mortality, and all the animals remained well during the entire period of observation. Two weeks after the original operation, reexploration was carried out to determine the fate of the grafts. All the grafts had taken well and appeared to be firmly attached to the liver surface. In areas where the skin had slightly overlapped liver capsule, cyst formation was beginning. Adhesions between omentum and graft were present in only four cases, no hæmatoma or subphrenic abscess was encountered, nor was there any evidence of persistent biliary drainage or fistula formation.

The animals were operated upon a third time at intervals of from four weeks to four months after the original liver surgery. The original operation was carried out again, the left lobe or the remaining middle lobe being resected, punctured with a trocar or torn manually. No effort was made to control the hæmorrhage, and all the animals rapidly became exsanguinated and died. After death, autopsies were performed, and microscopic sections of the grafted liver were taken. The grafts were intact, though greatly contracted. There was no evidence of metaplasia, nor had the grafts shown evidence of replacement by regenerating or abnormal liver tissue. Small cysts were observed wherever the graft had overlapped the parietal peritoneum or liver capsule. The practical results would appear to speak for themselves. It is to be hoped that they may be translated from their experimental setting into clinical practice.

[2] *Arch. Surg.*, November, 1954.

INTRAGASTRIC AND INTRADUODENAL ACIDITY.

ALTHOUGH it has been known for some time that the acidity and volume of gastric secretion, obtained on stimulation, are higher in patients with duodenal ulcer than in patients with gastric ulcer, published information about the acidity of the duodenum in these cases is meagre and often contradictory. Analyses of the contents have been made after short periods of fasting or after various types of test meal.

M. Atkinson and K. S. Henley[1] have determined the acidity of the contents of the ulcer-bearing part of the

[1] *Clin. Sc.*, February, 1955.

duodenum, that is, the first part, and of the stomach throughout the twenty-four hours under conditions which were as physiological as possible. Fifteen control subjects with no alimentary disease, fifteen duodenal ulcer patients and seven gastric ulcer patients have been examined. A double lumen tube was introduced into the stomach and the duodenum so that the duodenal apertures were in the first part of the duodenum, and the gastric apertures within the body of the stomach. The position of the tube was checked fluoroscopically at intervals not exceeding three to four hours between 8 a.m. and 10 p.m. After the introduction of the tubes five-millilitre samples were taken each hour for twelve to twenty-four hours. The subjects had a good standard diet spaced through the day. The pH of the samples was in most cases determined by a sealed glass electrode. The contents of the second part of the duodenum are seldom acid except briefly with the influx of duodenal contents. Free acid was found in the first part of the duodenum, under physiological conditions, in 70% of the daytime specimens and 64% of those taken during the night. In the patients with duodenal ulcer there was a high intragastric acidity and a high intraduodenal acidity during the day, but at night, while the gastric acidity remained high, the duodenal acidity fell to normal levels. An interesting and somewhat unexpected result was the finding that the mean level of gastric acidity was higher in the duodenal ulcer group and lower in the gastric ulcer group than in the controls, and all three groups showed a fall of acidity during the night. Intraduodenal acidity in the group with gastric ulcer was reduced during both day and night. Synchronous rises in intragastric and intraduodenal acidity occurred three to four hours after meals and the peaks in the intraduodenal acidity are related to increases in gastric secretion rather than to pancreatic and biliary factors. The coincidence of the peak levels of intraduodenal acidity and of pain in duodenal ulcer patients supports the commonly held view that acid is responsible for ulcer pain. The fact that the fluid bathing a duodenal ulcer is much more acid than that bathing a gastric ulcer raises interesting questions as to the part played by acid in the pathogenesis of the two conditions. An ulcer in the duodenum is also exposed to a highly acid fluid for much longer periods than one on the lesser curvature of the stomach.

A NEW METHOD OF DRUG ADMINISTRATION.

To describe "the tense, restless child who 'cannot sit still' and who bursts into a room like a small destructive whirlwind" L. C. Burket[1] uses the term hyperkinetic. It seems suitable, for children of this kind are certainly endowed with an excess of energy. Burket states that the hyperkinetic child is often characterized by an inability to concentrate for a reasonable length of time, by a tendency towards frequent outbursts of temper, and by impulsive, irrational behaviour. The hyperkinetic syndrome, as he calls it, may be psychic in origin or it may arise from brain damage caused by an accident or from a disease such as epilepsy, chorea or rheumatic fever. We may agree with this classification, but think that it is incomplete. Some children display excess of energy because it is "the nature of the beast". These children are naturally inquisitive. They cannot be put into any of the three groups mentioned by Burket. They notice more than other children do; they want to know what this is for and what that is for, why this happens and why that happens. We may describe these children as hyperkinetic, but they can hardly be included under any of the three groups which have been mentioned. This discussion about energetic children is perhaps beside the point deal with by Burket in his article. He states that these children often have to be given such drugs as phenobarbitone, and that it may be difficult for them to adhere to the rigid regimen required by long-term drug therapy. He reports that recently a new "sustained-release" phenobarbital capsule has become available. This capsule contains a great many minute phenobarbital pellets, some of which release the drug immediately, and the others are coated in such a way as to release small doses of the drug throughout the day. On the face of it, this seems to be a good idea, but, of course, its effectiveness will depend on the skill with which the preparation is prepared. It is something like the implantation of a drug under the skin so that it will act as a depot from which it is gradually absorbed. It is also something like the use of adrenaline in oil when the adrenaline is not absorbed immediately. Burket reports a study which he undertook on 38 hyperkinetic children—22 girls and 16 boys—ranging in age from two to twelve years. All had been under treatment with phenobarbital tablets for at least three months before the study was started. All these children were treated with the sustained-release capsule for two weeks, then by ordinary tablets for two weeks, and then again by the capsule for two weeks. During this six-week period, daily detailed progress reports were kept. In 24 of the 38 cases the result was excellent, the clinical improvement being greater than that achieved previously with the tablets; a good result was obtained in seven cases and a fair result in five. The result was poor in two cases because the children refused to take the capsules. It was found that in some cases a greater therapeutic benefit was obtained with the capsule with a lesser amount of phenobarbital. It certainly seems a good idea to have one medication in the day rather than several at stated intervals when both the child and the parent are likely to forget.

[1] *Am. J. M. Sc.*, January, 1955.

THE SURGERY OF CARDIAC ISCHÆMIA.

IN our issue of February 26, 1955, M. P. Susman, in the correspondence column, put in a plea for more study of the surgical treatment of cardiac ischæmia; it was disappointing that this important matter did not provoke discussion. Perhaps interest will be stimulated by an article in *The Journal of Thoracic Surgery* of January, 1955, in which Arthur Vineberg writes on four years' clinical experience with internal mammary implantation in the treatment of human coronary artery insufficiency including additional experimental studies. Vineberg has been working at this problem for over nine years, and the present paper gives the experimental evidence in support of the operation and its results in twenty-nine patients suffering from coronary artery insufficiency. Five of the patients died, fifteen are completely free of pain and some of them are working at hard physical labour. These results are encouraging, especially when it is remembered that after occlusion of one coronary artery less than half of the sufferers will survive five years. Vineberg emphasizes that there must still be a large amount of good myocardium remaining to receive the new blood supply; it is no good to attempt rejuvenation of fibrotic tissue in the heart any more than it is in other sites. Therefore operation must be performed as soon as possible once the indications are present, namely, (*a*) progressive coronary artery insufficiency without disability, (*b*) partial or total disability but without angina at rest. Contraindications to operation are: (*a*) evidence of left ventricular failure, (*b*) enlargement of the left ventricle unless the enlargement is due to moderate hypertension, (*c*) advanced essential hypertension, (*d*) recent myocardial infarction or active disease, (*e*) the occurrence of angina at rest. Operation is not advised for patients who have only occasional anginal pain on exercise or emotion, if they are able to work and live a fairly normal life. In the discussion that followed the presentation of this paper there was some adverse criticism as well as support. Several speakers pointed out that other operations may give results as good as Vineberg's. Samuel Thompson, of New York, for example, discussed the benefit to be obtained by inducing an inflammatory pericarditis by dusting the surface of the heart with magnesium silicate powder; he based his claims on 134 operations, with good results in 90%. Dr. Charles Ripstein doubted whether any of these operations gave any lasting benefit and is conducting a review of them. His findings should be carefully scrutinized when they are published.

Abstracts from Medical Literature.

MEDICINE.

Complement in Severe Proteinuria.

KURT LANGE AND EUGENE J. WENK (*Am. J. M. Sc.*, October, 1954) state that they have found previously that serum complement levels are low in the nephrotic syndrome but are normal in Kimmelsteil-Wilson's syndrome and in renal amyloidosis. The lowering of serum complement activity in the nephrotic syndrome is due to a low content of complement component C'2. In some cases C'4 is also depressed. Certain complement components are excreted into the urine in small amounts in all cases of severe proteinuria studied, irrespective of serum complement level, the hypoproteinæmia or the degree of proteinuria. The predominant complement component excreted into the urine differed and showed no relation to the type of disease or the degree of proteinuria. The amount of complement which could possibly be inactivated by this loss of one component is too small to explain the lowering of serum component in the nephrotic syndrome. An excretion of complement components of a similar magnitude does not lead to lowering of serum complement levels in Kimmelsteil-Wilson's syndrome or renal amyloidosis.

Generalized B.C.G. Infection.

H. THRAP MEYER (*Acta tuberc. scandinav.*, Volume XXIX, Fasciculus iii, 1954) reports the case of a young man who was vaccinated with B.C.G. A year later he developed an axillary abscess, which was incised and curetted, and the following year abscesses in the chest wall, the pus from which yielded tubercle bacilli of the B.C.G. type on culture. Three years after vaccination he suffered from serous meningitis. No microbes could be cultivated from the cerebrospinal fluid, but during his stay in hospital the B.C.G. was recovered from pus from thoracic sinuses. A few months later he began to show signs of tuberculous foci in the thoracic spine, in one kidney, in the sterno-clavicular joint, in the trochanteric region, in the pleuræ and in the lungs. The generalized tuberculosis was also shown to be caused by a strain of the B.C.G. The author states that the case is unique. The vaccine which caused the trouble was administered from the same bottle to five children, none of whom suffered any untoward reactions, and the vaccine from the same batch was issued to 128 other users, none of whom reported ill effects. Inoculation into animals gave normal results.

Migration of an Intravenous Catheter into the Heart.

D. D. TURNER AND S. C. SOMMERS (*New England J. Med.*, October 28, 1954) report a case in which a polyethylene catheter was inserted into the right median cubital vein for the purpose of an intravenous infusion in a patient suffering from infarction of the heart. It was secured with a silk ligature. It was also secured to the skin with adhesive tape. After two days, during which the patient was restless, the catheter, 22 centimetres (nine inches) long, disappeared. It could not be felt in the vein. The patient died of cardiac infarction two days later, and at autopsy the catheter was found partly (four centimetres) in the right atrium and partly (18 centimetres) in the superior *vena cava*. There was a mural thrombus where the catheter made contact with the atrial wall.

Treatment of Rheumatoid Arthritis by Adrenal Stimulation.

H. F. WEST AND G. R. NEWNS (*Lancet*, March 19, 1955) have studied a small group of rheumatoid patients receiving mild to moderate adrenal stimulation through the administration of corticotrophin for from one to two years. They state that the results of the study suggest that the maintenance of a daily urinary output of 20 to 35 milligrammes of 17-ketogenic steroids will favourably influence the course of moderately severe rheumatoid disease. In consequence, they consider that every effort should be made to extend this therapeutic trial and to find a more satisfactory adrenal stimulant for long-term use than the present preparations of corticotrophin. They state that the technical disadvantages of corticotrophin treatment lie in the need for daily injections and for urinary assays. Both disadvantages would be reduced if corticotrophin could be produced with a longer action and with a constant effect when given intramuscularly. The further disadvantage of corticotrophin is the possibility of allergic or anaphylactic reactions in patients given repeated courses. On the credit side is the fact that on withdrawal of corticotrophin therapy the adrenal glands are left active and possibly enlarged, whereas on withdrawal of cortisone treatment they are inactive and possibly smaller.

Treatment of Typhoid Fever with Tetracycline.

K. C. WATSON (*Lancet*, March 26, 1955) reports the results of treatment with tetracycline of six patients suffering from typhoid fever. He states that the response observed was poor and that for the treatment of this disease tetracycline seems to be considerably less useful than chloramphenicol.

Treatment of Pulmonary Sarcoidosis with Streptomycin and Cortisone.

C. HOYLE, J. DAWSON AND G. MATHER (*Lancet*, March 26, 1955) report the treatment of patients suffering from pulmonary sarcoidosis with streptomycin and PAS in 30 cases, and with cortisone in addition in 20 cases. They state that patients whose disease had been present for more than two years seldom improved during treatment with streptomycin and PAS, whereas those with a shorter history often improved. The time of onset of radiographic improvement and of its greatest extent was similar to that found with chronic pulmonary tuberculosis treated in the same way. Of the 12 patients who responded to treatment, three subsequently suffered relapse. When cortisone was given as well, 17 of 20 patients improved radiographically. Regression of sarcoid follicles in the liver was observed in eight patients who had serial biopsies. With additional cortisone therapy an improvement took place more rapidly than with streptomycin therapy alone, and was also more frequent and more complete among those suffering from recent disease. After treatment 11 patients suffered complete relapse and four suffered partial relapse. Prolonged treatment up to a year in duration was found to be safe and effective.

Nephrosis in Acute Pancreatitis.

M. J. LYNCH (*Arch. Int. Med.*, November, 1954) reports four cases of acute hæmorrhagic pancreatitis and one of focal pancreatitis in which pronounced nephrosis was a striking pathological finding. Two of the acute cases were investigated by frozen section fat stain technique, and widespread fat embolization was revealed. The author believes that fat embolism to the kidneys causes ischæmic atrophy of the tubules with replacement fibrosis. It is suggested that fat embolism or nephrosis is largely responsible for death in cases of acute hæmorrhagic pancreatitis; if recovery occurs, renal fibrosis may be the end result.

Prolonged Cortisone Therapy for Rheumatoid Arthritis.

J. J. BUNIM, M. ZIFF AND C. MCEWEN (*Am. J. Med.*, January, 1955) present data collected from observation over a four-year period of 78 patients suffering from rheumatoid arthritis (including nine suffering from juvenile rheumatoid arthritis) who were treated with cortisone for intervals varying from a few weeks to almost four years. From their study, the authors conclude that in properly selected cases of rheumatoid arthritis adrenal cortical steroids such as cortisone or hydrocortisone are very useful agents. The patients most suitable for hormone administration are those whose disease is severe, reversible, and of relatively recent development, but following a rapidly progressive, relentless course. It may also be useful for patients who have not responded well to, or are unable to tolerate, other anti-rheumatic drugs. Suitable subjects are patients who do not require larger than moderate doses of steroid for the control of their arthritis and who present no contraindications or unusual susceptibility to the side effects of the hormone. Many patients can tolerate maintenance doses of cortisone for several years without becoming refractory and without developing clinical symptoms of adrenal cortical insufficiency. The advantages of these hormones are that they produce a high rate of therapeutic response, especially in the first few months of treatment, and relatively early recovery of good functional capacity with restoration to employability and self-sufficiency, and that they aid in implementation of an effective rehabilitation programme. Most of the undesirable side effects are reversible, and many disappear even during continued administration. Certain limitations of

steroid therapy have become apparent during this study. These consist of failure appreciably to alter or arrest the extension of pathological processes of the disease, the frequent occurrence of relapses, at times quite severe when administration of the drugs is discontinued, and the development in some cases of serious complications or side effects of prolonged cortisone therapy.

E. C. TOONE, JUNIOR, AND R. IRBY (*ibidem*) report that at the end of three years 11 patients of an original group of 35 suffering from rheumatoid arthritis treated with cortisone have been benefited sufficiently to continue treatment with the drug. None of these patients suffered any serious adverse reactions. Five deaths occurred in the series, four of which were probably directly or indirectly the result of the cortisone administration. In one case, death occurred twenty months after administration of the drug had been discontinued. Adverse or toxic reactions of a nature and severity sufficient to require discontinuance of treatment developed in 10 cases. However, in two of these it was doubtful that the cortisone was entirely responsible. Minor adverse or toxic reactions were encountered often and in a variety of forms. Many miscellaneous clinical conditions developed in this group of patients in the course of cortisone treatment and were always a source of considerable apprehension and concern on the part of both the physician and the patient. Many of these had no direct or indirect relationship to the treatment, and it was important to evaluate the situations correctly. The authors conclude that cortisone has a definite but limited use in the maintenance management of rheumatoid arthritis. It should not be used routinely, but only after other measures have been adequately tried.

Pulmonary Embolism.

A. TOWBIN (*J.A.M.A.*, September, 1954) reviews pulmonary embolism as a cause of death in institutional patients. He states that he found *post mortem* massive pulmonary embolism as the cause of death in 14% of patients. Occlusion of swollen pulmonary arteries occurred in 11·5%, mainly in the lower lobe of the right lung. Sudden death occurred in 18%, and was often diagnosed clinically as due to coronary occlusion. The signs in thrombo-embolic lung lesions were often equivocal. These conditions were most frequent in old age. In many cases, death occurred slowly, with signs frequently attributed to bronchopneumonia.

The Use of Reserpine in the Hypertensive Arteriosclerotic Syndrome.

J. KLEH AND J. F. FAZEKAS (*Am. J. M. Sc.*, November, 1954) evaluate the effect of reserpine in the treatment of the hypertensive arteriosclerotic syndrome, the study being based upon eight subjects. These had sustained diastolic hypertension and overt manifestations of cerebral vascular disease, and all had experienced single or multiple episodes of cerebral thrombosis; five of the eight were mentally deteriorated. They had also dizziness, weakness in the legs, headaches, staggering and visual disturbances. Studies of cerebral hæmodynamics and metabolism were made upon these patients. After the baseline hypertensive readings were evaluated, treatment was begun with 0·4 milligramme of "Serpasil" daily in divided doses; then the dose was increased to 1·0 milligramme after four days, and increased by 0·25 milligramme at weekly intervals to maximum blood pressure depression. In some cases the dose was increased still further. The patients were maintained for at least four weeks on the maximum dose schedule. A moderate reduction of blood pressure was noted with a daily dose of 1·0 milligramme of the drug. At higher doses, while there was in several cases an increased hypotensive effect, there was also a concomitant decrease in the patients' responsiveness. If the dose was further increased, no appreciable change in blood pressure occurred, but lethargy increased. The maximum blood pressure depression was achieved with a dosage of from 1·5 to 3·0 milligrammes per day and averaged 24 millimetres of mercury, systolic, and 27 millimetres, diastolic. The maximum dose administered was 4·0 milligrammes daily. There was no lessening in any of the signs or symptoms attributed to vascular insufficiency in any of the patients at the dosage levels studied. Nasal congestion and diarrhœa offered no particular problem, and there were no significant changes in the hæmogram, blood urea nitrogen level or electrocardiogram. "Serpasil" had no material effect on either cerebral hæmodynamics or metabolism. The authors conclude that this drug alone in the dosage employed is of little value in the treatment of the hypertensive arteriosclerotic patient, although it is noted that it produced a significant depression of the average blood pressure in all subjects.

Myasthenia Gravis.

R. S. SCHWAB AND W. H. TIMBERLAKE (*New England J. Med.*, August 12, 1954) describe the treatment of *myasthenia gravis* with pyridostigmin ("Mestinon"). They state that neostigmine is the drug of choice for this condition. Its disadvantages when given by mouth are its brief action, the fact that it must be repeated every two or three hours, and its effect on the gastro-intestinal tract, causing cramps and diarrhœa. Atropine relieves these symptoms, but it may cause confusion and may mask the action of neostigmine, causing overdosage with consequent muscular weakness. Pyridostigmin is an analogue of neostigmine bromide. It has been said to have a prolonged effect, but the authors did not observe this action at first (in 1948). In 1953, larger doses were given, 60 milligrammes in place of 15 milligrammes of neostigmine. The drug was effective, but had no prolonged action. It did not cause unpleasant gastro-intestinal symptoms. In some cases it was not so effective as neostigmine.

Infections During Chemotherapy.

L. WEINSTEIN, M. GOLDFIELD AND TE-WEN-CHANG (*New England J. Med.*, August 12, 1954) discuss infections occurring during chemotherapy. After treatment with penicillin *Hæmophilus influenzæ*, *Escherischia coli*, *Aerobacter aerogenes*, *Pseudomonas æruginosa*, *Neisseria*, *Proteus vulgaris*, *Klebsiella pneumoniæ* and monilia may become apparent or even predominant. Generally, cessation of treatment has led to re-establishment of the normal pharyngeal flora. With other antibiotics other organisms may flourish in the upper part of the respiratory tract. Chlortetracycline, chloramphenicol and oxytetracycline produced a predominantly Gram-negative flora, whereas broad-spectrum drugs produced a change less frequently. Moulds and yeasts in the mouth change during treatment with most chemotherapeutic agents; and similar changes have been observed in the intestinal tract, ear, bowel, bladder and lungs. A close watch on the bacteria of the affected area is advised.

Hydralazine.

I. A. FEDER (*New England J. Med.*, August 12, 1954) discusses the treatment of hypertension with hydralazine ("Apresoline"). Severe toxic effects have been reported, including early rheumatoid arthritis and acute systemic *lupus erythematosus*. A case is reported in which 25 milligrammes of hydralazine were given four times a day. The dose was later increased to 800 milligrammes a day. The blood pressure fell from 240 millimetres of mercury, systolic, and 150 millimetres, diastolic, to 175 millimetres, systolic, and 120 millimetres, diastolic, and eventually to 165 millimetres, systolic, and 110 millimetres, diastolic. One year later the patient developed a thrombus in the leg, fever and anorexia with albuminuria. Raised reddish lesions appeared on arms and hand. Generalized muscular pains, weakness, anorexia and fever continued. Hydralazine was stopped; the blood pressure rose to 230 millimetres of mercury, systolic, and 150 millimetres, diastolic. ACTH, 25 milligrammes in 500 millilitres of 5% glucose solution, was given intravenously over twelve hours with relief. This was replaced by 200 milligrammes of cortisone daily with gradual reduction over four weeks. Great benefit and eventual recovery followed this treatment.

Results of Mitral Commissurotomy.

F. H. ELLIS, J. W. KIRKLIN, R. L. PARKER, H. B. BURCHELL AND E. H. WOOD (*Arch. Int. Med.*, November, 1954) report the clinical and hæmodynamic results following surgery for mitral stenosis in 131 patients. The mortality rate was 8·4%, but of the survivors 87·2% achieved an excellent result or were significantly improved. A significant factor influencing post-operative results was the anatomical status of the mitral valve; 43·2% of patients with scarred, immobile or calcified valves either died subsequently to operation or were unimproved. During operation the left atrial and pulmonary artery pressures fell, and three weeks after operation it was shown in addition that the cardiac output increased significantly with exercise. These changes persisted, and there was often improvement a year or more after operation. These objective measurements were in harmony with the clinical improvement.

Medical Societies.

PÆDIATRIC SOCIETY OF VICTORIA.

A MEETING of the Pædiatric Society of Victoria was held at the Royal Children's Hospital on Wednesday, February 9, 1955.

Addisonian Crises in the Adreno-Genital Syndrome.

DR. ELIZABETH TURNER described the case of a baby with adreno-genital syndrome who developed an Addisonian crisis at eleven days of age. The baby, who was the second child of a healthy mother, was treated at the Queen Victoria Memorial Hospital. The pregnancy was normal, and the baby was born normally, at full time, and weighed six pounds six ounces. At birth she was found to have a greatly enlarged clitoris with the opening of the uro-genital sinus on its inferior aspect. For the first few days she gave no cause for worry, but on the third day she became jaundiced with puffy eyes. By the fifth day the weight had dropped to five pounds thirteen ounces, and breast feedings were complemented; but by the eighth day the weight was down to five pounds eleven and three-quarter ounces. The baby took feedings very slowly, and on the eleventh day started to vomit small amounts. Bowel actions were normal, and urinary output seemed satisfactory. During the next twenty-four hours the baby developed a dusky hue and became irritable. The skin was dry and hydration poor. The feeding strength was weakened, and the apparatus for a subcutaneous infusion of glucose with Hartman's solution was set up. The baby became increasingly drowsy and cyanosed, and developed rapid irregular respirations with marked rib retraction. During the next few hours she was in a collapsed condition with a cold clammy skin, and was vomiting small amounts of greenish fluid. She appeared to be showing all the signs of acute adrenal insufficiency.

The serum sodium content at this stage was 290 milligrammes per 100 millilitres, and the blood sugar content 120 milligrammes per 100 millilitres. The electrocardiogram showed potassium deficiency. The baby was immediately given normal saline, cortisone and DOCA by the intravenous route, vitamin *K* by the intramuscular route and glucose syrup on the tongue half-hourly. During the next twelve hours the condition improved, vomiting ceased and the greyish coloration disappeared leaving a bronze pigmentation. After twenty-four hours oral feedings were taken eagerly. The intravenous drip administration was discontinued, and salt was added to the oral feedings up to three grammes per day. Treatment with one milligramme of DOCA and ten milligrammes of cortisone per day was continued.

The baby progressed well. After two and a half months the administration of DOCA was suspended, and the pigmentation began to fade. After a further three weeks cortisone therapy was discontinued, but within twenty-four hours the baby was less eager for feeds and vomited small amounts. There was some weight loss, and the skin was dry and greyish. She had frequent twitchings and jumped when disturbed. An intravenous infusion of half normal saline with 5% of glucose and ten milligrammes of cortisone was commenced, and after recovery the cortisone, DOCA and salt in feedings were continued as before. At three and a half months two 100-milligramme pellets of DOCA were implanted in the subcutaneous fat of the *mons pubis*. Progress to date had been satisfactory. The infant was now thriving and accomplishing normal weight and height increments on ten milligrammes of cortisone by mouth daily, and three and one-third grammes of salt, and it was proposed to repeat the DOCA implants each three months. The masculine features of the genitalia were receding.

Dr. Turner then discussed briefly the theories of ætiology of the condition and rationale of treatment. She said that once such an infant had been born to a mother she had a one-in-four chance of having another infant similarly affected. There seemed to be two theories of the ætiology: first, the inheritance of a defective gene and, second, a maternal imbalance acting at a very early stage of embryonic development and producing an abnormal adrenal cortex.

Apparently the adrenal cortex had difficulty in converting the precursor steroid 17-hydroxy-progesterone into compound F, the carbohydrate-regulating steroid of very similar structure to cortisone. Instead the pathway of biosynthesis was diverted to the production of abnormal steroids which had androgenic activity. To meet the requirements of compound F, the anterior pituitary lobe secreted excess ACTH, which whipped up the adrenal cortex to hyperplasia and the production of more compound F, but at the same time produced an increase in androgens and the consequent masculinization of the fœtus. This androgen had an inhibitory action on the anterior pituitary lobe suppressing follicle-stimulating hormone, and the ovary therefore did not produce sufficient œstrogen to bring about breast development or menstruation. The administration of cortisone relieved the strain on the whole system. ACTH production was reduced, and adrenal cortical hyperplasia diminished. Compound F and cortisone were sufficient to regulate the carbohydrate mechanism, and follicle-stimulating hormone stimulated the ovary to produce œstrogen with consequent feminization. The Addisonian-like crisis which proved that adrenal cortical abnormality was present clinched the diagnosis of adreno-genital syndrome without further investigation.

The Addisonian crisis itself was not simply an electrolyte disturbance due to deficiency of the salt hormone from the *zona granulosa*, but was apparently a much more complex disturbance involving the presence of desoxycorticosterone antagonists which aggravated the salt-losing tendency.

The rapid appearance of bronzing of the skin was a phenomenon which Dr. Turner said she did not clearly understand. She wondered whether Dr. Brian Hudson could throw some light on it during the discussion.

Dr. Turner said that an attempt had been made in the present case to instil "Lipiodol" into the infant's uterus through the cloacal opening and to take an X-ray picture, but without success. There was probably no connexion between the vagina and urethra in the present case, although in the commonest abnormality there was.

DR. M. J. ROBINSON described the case of a baby who was aged thirteen days when admitted to hospital. The baby's birth had been normal, and the pregnancy uneventful. The birth weight was seven pounds two ounces, and breast feedings were taken well, the birth weight being regained on the ninth day. The baby was originally regarded as a male. Four days before admission to hospital the baby started to vomit about once a day in a projectile fashion, and became lethargic and disinclined to feed.

The pregnancy was the mother's third. The first child, a boy, was now two and a half years old and healthy. The second child was said to be a hermaphrodite and had died in Perth at the age of one month. The Children's Hospital, Perth, later provided the information that the child had been a female pseudohermaphrodite who died in an Addisonian crisis. At autopsy a normal uterus, Fallopian tubes and ovaries, and bilateral adrenal hyperplasia had been found.

On examination on the day of admission to hospital the baby was rather drowsy and not obviously dehydrated, but the body was definitely pigmented. The genitalia appeared abnormal, in that the clitoris was grossly hypertrophied with the orifice of the uro-genital sinus located at the tip of the glans. *Labia minora* were not present and the *labia majora* were fused. Thus the appearance was very suggestive of hypospadias with bilateral cryptorchidism. No testes could be palpated in the perineum or the inguinal region. By next morning the baby's condition had becomc much worse. Dehydration out of proportion to the amount of fluid that could have been lost by vomiting was present, and the peripheral circulation was poor. The heart sounds were scarcely audible, and the pulse rate was 85 beats per minute.

Serum electrolyte levels had been estimated on the previous day with the following results: sodium 120 milliequivalents, chloride 83 milliequivalents, potassium 9·8 milliequivalents and bicarbonate 28 milliequivalents per litre. An electrocardiogram taken at this stage showed a slow ventricular tachycardia with absence of *P* waves and grossly prolonged *QRS* complexes, changes such as are seen in an extreme hyperkalæmia.

The diagnosis of an Addisonian crisis in a female pseudohermaphrodite was made and treatment begun. The calculated fluid loss was remedied by intravenous replacement with 2·5% glucose in half isotonic saline, and at the same time cortisone was administered, 25 milligrammes being given twelve-hourly intravenously on the first day.

It was felt that the high potassium level might prove fatal before the electrolyte balance, and in particular the sodium level, could be restored. Dr. Robinson said that the administration of glucose and insulin, as a temporary means of mobilizing potassium into cells, had often been used by others in reducing very high serum potassium levels in patients with acute renal failure, until exchange resins took effect or dialysis could be carried out. Therefore the baby

was given two units of regular insulin subcutaneously and five grammes of glucose intravenously. The known sensitivity of patients in an Addisonian crisis to insulin caused some worry, but although sweating started within half an hour after the insulin, this was controlled with a further five grammes of glucose given intravenously.

Four hours after treatment had been commenced, an electrocardiogram showed that the cardiac rhythm had returned to normal, and the *P* and *QR* intervals were normal, but the complexes were still of low voltage. On the next day, despite 35 ounces of fluid given intravenously, the hydration was not absolutely satisfactory, but serum electrolyte levels were improved. However, the potassium level was still high, being 8·2 milliequivalents per litre. Intravenous therapy was discontinued at the end of the second day, and fluids were given orally. The baby received three ounces of fluid each three hours by gavage, and four grammes of sodium were added to the daily feeding. Cortisone was reduced to 25 milligrammes once daily, and after one week this was further reduced to 12·5 milligrammes daily.

Electrocardiography was repeated several times, but the findings remained normal. Serum electrolyte estimations showed a progressive rise of sodium and chloride levels to normal and a fall in potassium level. With a combination of five milligrammes of cortisone daily and up to six grammes of sodium chloride added to feedings, the baby maintained its weight but did not gain in weight. Approximately ten days after admission to hospital daily administration of two milligrammes of DOCA by intramuscular injection was commenced and the added sodium chloride reduced to three grammes daily. The baby immediately began to gain in weight, and the weight gain was maintained.

Urinary ketosteroid estimations performed both during the administration of cortisone and when cortisone was withheld for one week showed the level of excretion to be below one milligramme in the twenty-four hours.

Dr. Robinson said that the case showed how infants with adrenal hyperplasia were very liable to develop sodium depletion and dehydration—namely, an infantile Addisonian state. The reasons for that still did not seem to be clear, although several theories had been advanced, some of which had been discussed by Dr. Turner.

The principles of therapy in such cases appeared to be, first, the immediate replacement of sodium and water, and in a crisis that would have to be effected intravenously. Milder grades of sodium depletion could be corrected by the addition of sodium chloride to the feedings. Desoxycorticosterone acetate might also be required. At the same time it appeared important to depress androgen secretion by cortisone, which acted via the anterior pituitary lobe. But cortisone was a poor salt-retaining hormone, and many patients required DOCA as well as additional salt and cortisone.

The extremely high level of serum potassium seen in the present case was not commonly observed, but some elevation of serum potassium level was always present with deficient mineralocorticoid secretion. Sodium replacement alone might adjust the high potassium level (Bunge phenomenon), but in the particular case under consideration it was felt that the time factor was vital.

DR. S. J. WILLIAMS opened the discussion and, referring to the second case, emphasized the familial nature of the condition and the distress of the mother of the child, when he had told her that the second child was a hermaphrodite. The child was then one day old. The mother realized the danger of a sudden crisis after her experience of the previous child, which died after an Addisonian crisis.

Dr. Williams considered that such infants probably needed to be observed in hospital for at least three months after birth. Even in hospital a close watch was required for the sudden appearance of the features of the Addisonian crisis.

DR. B. HUDSON said that his experience of the Addisonian crisis had been confined to adults, and he had no experience of the event associated with the adreno-genital syndrome. However, he was very interested to note that DOCA had not been exhibited at the onset of treatment, and therefore apparently the mineralocorticoid in cortisone must have been sufficient at first, but not for long. The adrenal crisis seen in children differed in some respects from that seen in adults. It was unusual in the latter to find marked alteration in electrolytes. He had never seen the serum potassium level above 5·5 milliequivalents per litre. He wondered what the blood sugar level had been in Dr. Robinson's case.

Dr. Hudson went on to say that compound F for intravenous administration was now available in Australia. Also, for maintenance therapy with DOCA some people preferred to use the injectable long-acting desoxycorticosterone trimethyl acetate instead of pellets. The preference was a personal one, but many patients needed extra mineralocorticoid during the summer months. He wondered whether later on it might be possible to remove the adrenal and maintain the patients with electrocortin.

Regarding Dr. Turner's comment about pigmentation in the patients, Dr. Hudson thought that it was added evidence of excessive ACTH activity. The cause of pigmentation in Addison's disease probably had its origin in the pituitary gland. A melanophore-expanding hormone had been extracted from fresh human hypophysis and also from the blood of a patient with Addison's disease. Pigmentation was thought to be due to the over-activity of the pituitary melanophore-expanding hormone.

Dr. Robinson, in reply, said that the blood sugar level in his case had been 150 milligrammes per 100 millilitres of blood. Other workers had said that the blood sugar levels in infants with the condition were normal. However, all serum potassium levels had been raised.

DR. H. E. WILLIAMS mentioned a patient who had been under the care of his unit and had failed to make satisfactory growth when treated with cortisone and salt only. The introduction of DOCA resulted in growth improvement. During a respiratory infection the baby rapidly developed a "crisis", and the dose of DOCA had to be increased. She had been kept on all three therapeutic aids for two years now. Dr. Williams said that Lawson Wilkins recommended a daily dose of 10 to 12 milligrammes of cortisone. The patient referred to was having 12 milligrammes per day, but that had been increased to 17·5 milligrammes per day recently, as the ketosteroid excretion was rising.

DR. J. COLEBATCH then referred to a baby who had been under his care at the Women's Hospital. It was the second child in the family, the other one being normal. The birth weight was six pounds twelve ounces, and the baby appeared well and remained so. However, a genital deformity was present, in that the clitoris was enlarged and the labia were rugose. Dr. Colebatch showed "Kodachrome" slides to demonstrate the abnormality, and to show that at two and a half months and seven months of age there was still some clitoral enlargement. He asked whether any advice could be offered regarding the future management of the baby.

DR. P. TAFT commented on Dr. Colebatch's case and said that babies like that had been described, and in some instances there was evidence of excessive androgen excretion in the mother. One had had an adrenoblastoma.

Malrotation of the Duodenum with Special Reference to "Cyclical Vomiting".

DR. D. STEPHENS gave a paper on malrotation of the duodenum with special reference to cases of "cyclical vomiting". He said that malrotation of the mid-gut was a well-known abnormality which caused duodenal obstruction in the newborn. The diagnosis was made and confirmed by appropriate operation, which readily rectified the condition. The abnormality might be present but escape notice in the neonatal period or produce only mild symptoms. It might then predispose to attacks of bile vomiting or painful episodes during childhood. It was the detection of those cases with an organic basis for the symptoms from the great group of cases of recurrent vomiting and pain of non-surgical origin in children that he wished to discuss.

In children who presented in a crisis of acute duodenal obstruction with or without a previous history of minor or major episodes the condition was easily diagnosed at operation and did not present a very difficult problem. But those who had crises of short duration, often recurrent or cyclical, presented diagnostic enigmas. The diagnosis must then be made in the symptom-free phase.

That was all the more important when one saw that vomiting attacks had occurred in some children for nine years undiagnosed.

The special types of malrotation which were discussed in the paper and which caused the obstructive symptoms affected the mid-gut. Those which involved other organs in conditions such as *situs inversus abdominalis* and *totalis* were excluded.

Dr. Stephens said that he had been able to study 12 cases of malrotation of the mid-gut, which had presented in children between the ages of five months and nine years. Eight of the cases were studied at the Hospital for Sick Children, Great Ormond Street, London, and four at the Royal Children's Hospital, Melbourne. He wished to acknowledge his thanks to the physicians and surgeons of both

those hospitals for permitting him to study their cases. In considering the clinical pattern in all the cases, vomiting, sometimes copious, was a prominent symptom. In ten other children, the vomiting was cyclical at intervals varying between three weeks and three months. The duration of the attacks ranged from a few minutes to several days. In eight the vomitus was definitely green, and in four the colour was not recorded. The vomiting turns occurred for an average of thirty-three months before definitive treatment was administered, though one child presented on the first occasion in an obstructive crisis, and in another the attacks had recurred every few months for nine years. In eight, vomiting was the presenting symptom.

Abdominal pain occurred in seven children, but in one only was it the presenting symptom, though in two appendicectomy had been embarked upon at the time of the crisis.

One baby first presented with a feeding difficulty and another with attacks of vomiting and frequent fluid motions.

During an obstructive attack the infant or child appeared hollow-eyed from dehydration. The abdomen was flat or scaphoid in all except one case, and in some cases tenderness was elicited slightly above and to the right of the umbilicus.

When examined between attacks, the child appeared healthy and symptom-free.

Dr. Stephens then discussed the embryology of the condition. He said that the mid-gut comprised that portion of the alimentary tract which lay between the third part of the duodenum and the middle of the transverse colon. During development of the mid-gut, elongation took place, mainly in the segment proximal to the Meckel's diverticulum. The intestines, suspended on the dorsal mesentery and the superior mesenteric vessels, emerged through the umbilicus into the body stalk. Later they returned through the umbilical opening and disposed themselves around the root of the vessels, the duodenum coming to lie behind and the colon in front, the rotation taking place in an anti-clockwise direction. The small bowel became arranged on the left and the proximal half of the colon to the right.

Dr. Stephens said that Dott (1923) had described and illustrated clearly most of the irregularities in rotation of the mid-gut, and it was from his article that the colour photographs shown during presentation of the paper were taken.

Malrotations occurred in two main types: (*a*) non-rotation, in which the duodenum and jejunum returned and stayed on the right side, though lower coils lay on the left; (*b*) reversed rotation, in which the duodenum followed by the jejunum crossed over to the left in front of the superior mesenteric vessels, and the caecum and ascending colon together with the small segment of ileum distal to the Meckel's diverticulum might become wrapped around the straight stem of the fourth part of the duodenum. That volvulus was formed during development at about the tenth week of intrauterine life. Crises of obstruction in the bases reviewed were due not to formation of the volvulus, but simply to a few degrees of tightening of the twists. That was clearly seen when operation was performed in the quiescent phase, when the volvulus was fully formed but was creating no symptoms.

Both in (*a*) and in (*b*) the caecum might lie under the liver or descend to the iliac fossa. The caecum and ascending colon were freely mobile, but gained a short-based or, less often, a long-based attachment to the posterior abdominal wall. The mesentery lay like a veil across the duodenum, and in the case of the volvulus tightened on the duodenum enhancing the obstruction.

The obstruction occurred at the junction of the third and fourth segments of the duodenum, which was the junction of the fixed and mobile parts of the abnormally directed duodenum. The change in direction caused an acute folding on itself of the duodenum. This became more marked and obstructive as a result of tension of the rudimentary mesentery of the floating colon or caecum. In the volvulus of reversed rotation, the duodenum was obstructed chiefly by the tighly drawn mesentery, but also by the wrapping of the colon around the duodenum.

It was that abnormal change in direction of the fourth part of the duodenum which, when studied radiographically, was the chief diagnostic sign.

In discussing the radiographic features, Dr. Stephens said that barium meal studies demonstrated the size and course of the stomach and duodenum.

The normal duodenum was "C"-shaped and of uniform calibre. The lower part of the "C", corresponding to the third part of the duodenum, crossed the body of the third lumbar vertebra and continued cranially as its fourth part along the left border of this vertebra and the one above to complete the "C".

In the obstructive phase the diagnosis was easier. The duodenum filled to larger than normal dimensions to the point of obstruction, where the lumen became occluded. Little or no barium passed into the collapsed deflated small intestine. The barium lay to the right of the left border of the vertebral column. The fourth part of the duodenum was invisible.

In the non-obstructive phase, special efforts had to be made to follow the bolus of barium as it was propelled through the whole course of the duodenum. That might be time-consuming, as there was often a temporary hold-up proximal to the site of change in the direction of the fourth part. The course taken by the duodenum in those conditions resembled the letter "S", with the fourth part completing the lower curve of the "S" below the third lumbar vertebra.

In the non-rotation group, the duodenum and proximal part of the ileum lay to the right of the left border of the spine. The fourth part then coursed downwards instead of upwards, and emptied its content of barium into the jejunum, the upper coils of which lay towards the right side.

In the reversed rotation with volvulus, the fourth part of the duodenum was constricted slightly and took a downward and anterior course. At the points of constriction the fourth part was lifted off the vertebral bodies by the air-containing coils of the colon or terminal part of the ileum which were wrapped around it.

If further confirmation was required, a barium enema examination might show in some cases a malplaced caecum or partial occlusion of the lumen of the transverse or ascending colon at the site of the volvulus. It was emphasized that malposition of the caecum was more common without malrotation of the remainder of the mid-gut than with it, so that finding alone was not diagnostic.

Regarding treatment, Dr. Stephens said that, the cases having been selected by radiography for operation, the technique used was that described by Ladd (1933). The main principles were: (i) to unwind the volvulus, if present, in a clockwise direction, the status being reduced from reversed rotation to non-rotation of the duodenum, (ii) to divide the rudimentary mesocolon where it crossed the duodenum and clear the folded part of the fourth part of the duodenum to allow it to lie on the right side without kinks, and allow the colon to rest without fixation on the left side—that left a moderately large deperitonealized area between duodenum and colon, and (iii) to remove the appendix.

His results in the twelve cases showed that two patients died in the crisis of obstruction; one died within a few hours after an appendicectomy operation, and one, a mongol, died with the condition undiagnosed.

Nine were operated on for relief of obstructive symptoms, either in a major crisis or in the intervening quiescent phase. All were relieved of their symptoms, but two returned with intestinal obstruction due to bands arising from the deperitonealized area. One of those two patients was relieved by operation, and one died within a few hours of operation.

One child, whose main symptom was diarrhœa, had been improved considerably, but the final results could not be assessed, as operation had been too recent.

Unfortunately the follow-up was not complete, but the average duration of good health subsequent to operation in ten children was twenty-nine months, the longest being six years and eleven months. The results of the operation were highly satisfactory as judged from other reports in the literature.

To conclude, Dr. Stephens made the following statements. (i) Malrotation of the duodenum was a rare but important basis for attacks of intermittent vomiting which were amenable to surgical correction. (ii) Twelve cases had been studied, exclusive of the neonatal group. (iii) The fourth part of the duodenum on barium radiography was the key to the diagnosis; the duodenum was "C"-shaped normally, but in malrotation of the mid-gut it was "S"-shaped. (iv) Ladd's operation was the operation of choice in alleviating the symptoms.

DR. V. L. COLLINS opened the discussion and said that the importance of the paper was that it showed that a group of the so-called cyclical vomiters had a surgical basis for their symptoms. However, the condition was rare. Gross's figures of the age at which children with malrotation came to surgery showed that 59% presented in the first year. Only

a small percentage gave trouble later, and some of those would present with acute vomiting and obstruction with no preceding history. Therefore only a few were left to present the syndrome of cyclical vomiting.

Dr. Collins's attention had first been drawn to the condition by the case of an adult of twenty-one years who had had bouts of vomiting. Operation revealed the condition. Then he had seen a child of three years who had had about twelve attacks of vomiting, the last one being associated with the symptoms and signs of intestinal obstruction. However, he thought that the difficult thing to decide would be which child one would investigate to exclude the condition.

DR. R. SOUTHBY thanked Dr. Stephens for pointing out the surgical basis of some cases of cyclical vomiting. He had seen other surgical conditions which presented occasionally in a similar fashion, such as an undetected diaphragmatic hernia or strangulated internal hernia. He wondered whether there was also a group of cyclical vomiters with a psychiatric basis for their condition.

DR. W. RICKARDS said that no proper statistical study of a series of cases of cyclical vomiting had been carried out. A psychiatric diagnosis in such a case was only one of exclusion. Whilst he had been at the psychiatric clinic there had been only two patients referred for cyclical vomiting. However, many children presenting behaviour problems had infantile vomiting.

DR. H. HILLER said that he found it very difficult to follow the fourth part of the duodenum during the radiological examination of a barium meal.

DR. K. HALLAM agreed that the precise radiological examination of the anatomical abnormalities of the duodenum was difficult.

Dr. D. Stephens, in reply, said that he would limit the patients who received radiological examination to those who exhibited copious green vomitus with dehydration.

British Medical Association News.

NEW SOUTH WALES BRANCH NEWS.

ON April 20, 1955, the Council of the New South Wales Branch arranged a dinner in honour of Sir Charles Bickerton Blackburn which was held at the Hotel Australia, Sydney. The 160 members of the Branch who attended included many senior members of the profession. The chair was taken by Dr. H. Hastings Willis, President of the Branch. The toast of Sir Charles Blackburn's health was proposed by Sir Archibald Collins, President of the Federal Council of the British Medical Association in Australia.

Sir Archibald Collins traced Sir Charles Blackburn's career from the days when, as a medical student, he had left Adelaide because of an acute dispute in which the University of Adelaide was involved, and had chosen Sydney as his future medical home. This, Sir Archibald declared, was an extremely fortunate decision for Sydney. Prior to leaving Adelaide Blackburn had graduated in Arts and he had gained his baccalaureate in medicine and mastership in surgery at Sydney in 1899. On graduation he had become a resident medical officer at the Prince Alfred Hospital (it had not then attained its "Royal" status) and in 1901 had become medical superintendent. This had begun a most significant association which had lasted to the present time. Blackburn had become in turn honorary assistant physician, in-patient physician, and consulting physician. He had been a notable teacher and had left a lasting impression on the minds of generations of students. Sir Archibald Collins then referred to Sir Charles Blackburn's work as a practising physician and as a consultant and declared that there had been few, if any, like him. He mentioned Blackburn's doctorate in medicine, his activities in connexion with The Royal Australasian College of Physicians (with which Dr. C. G. McDonald would deal later) and his election to the Fellowship of the Royal College of Physicians of London and to the honorary Fellowship of the Royal College of Physicians of Edinburgh. Sir Archibald Collins then turned to the part which Sir Charles Blackburn had played in the affairs of the British Medical Association, New South Wales Branch. He had been a member of the Council continuously since 1910, and President in the year 1920-1921. His contributions to Council discussions were marked by knowledge, insight and wisdom. In the two World Wars Blackburn had served with distinction. In the first World War he had been twice mentioned in dispatches and had received the O.B.E., and his work at Number 12 Australian General Hospital in Egypt had been most outstanding. Sir Archibald Collins spoke of Sir Charles Blackburn's services to the University of Sydney as a member of the Senate and as Chancellor. He spoke of the long line of Chancellors and thought that Sir Charles Blackburn was preeminent among them. In conclusion, Sir Archibald Collins hailed Sir Charles Blackburn as the leader of the profession in Australia and assured him that those present delighted to do him honour and regarded him not only with high esteem but with affection.

Dr. C. G. McDonald supported the toast and said that as President of The Royal Australasian College of Physicians he had first-hand knowledge of all that Sir Charles Blackburn had done for that body. He had taken a leading part in the Association of Physicians, and when it developed into The Royal Australasian College of Physicians he had been the obvious and unanimous choice as its first President. He had presided at the inaugural ceremony in 1938 and Dr. McDonald thought that much of the success of the College, both in its fundamental basis and in its subsequent career, had been due to Sir Charles Blackburn's conversations with the Royal College of Physicians of London. That College had given the most helpful advice to the young Australasian body and had been friendly and helpful through the years. Dr. McDonald then spoke of Sir Charles Blackburn's association with the University of Sydney. He had been a Fellow of the Senate since 1919, Dean of the Faculty of Medicine from 1932 to 1935, and Chancellor since 1941. As Chancellor his activities had been characterized by charm and dignity. Dr. McDonald thought that Sir Charles was possibly one of the greatest Chancellors the university had known. One of the outstanding attributes manifested by him was the old Roman quality of *gravitas*, described by Matthew Arnold as higher seriousness. It was because of all Sir Charles Blackburn's exceptional qualities, which endeared him to all of them, that they had gathered together in his honour. Dr. McDonald echoed every word which Sir Archibald Collins had spoken.

The toast was drunk with musical honours.

Sir Charles Blackburn, who seemed to be considerably affected by the spirit of the occasion, began his reply by saying that an excuse for the gathering was the fact that he had attained his eightieth birthday. He deeply appreciated the generous remarks that had been made about him, but said that he was reminded of a tale about the funeral of a Negro which was attended by his widow. The parson had become eloquent about the virtues and good deeds of the deceased to such an extent that the sorrowing woman had pulled at the coast of the effusive speaker, saying in a loud whisper: "Parson, are you sure you're not burying the wrong man?" Sir Charles Blackburn went on to say that a great deal of any success that he had had was due to luck. Luck played a large part in the life of everyone, and it seemed to him that he had more than his share of it. He told some stories of his early life to illustrate this. He had, throughout his life, been fortunate in his colleagues and had enjoyed working with them. He insisted that he had derived most pleasure and satisfaction from the work for which he had not been paid. He thanked Sir Archibald Collins and Dr. McDonald and all who had come together that evening to do him honour.

VICTORIAN BRANCH NEWS.

THE June meeting of the Victorian Branch of the British Medical Association, at which Major-General F. Kingsley Norris and other medical officers of the Australian Military Forces will show films and speak on some medical aspects of atomic warfare, will be held on Wednesday, June 15, instead of the first Wednesday of the month.

Obituary.

CHARLES JAMES MARTIN.

IN the ranks of those who have had a profound influence on British medicine in general and Australian medicine in particular the late Sir Charles Martin has a prominent place. In England, as Director of the Lister Institute, he carried out and directed investigations of the most far-reaching importance, and in Australia he was a member of the teaching staff of the Universities of Sydney, Melbourne

and Adelaide in turn. In these universities he created a Martin tradition and even today he is spoken of in terms of respect and affection. He was, in fact, one of the founders of medical research in this country.

Charles James Martin was born in London in 1866 and he received his medical education at King's College and at St. Thomas's Hospital, London. He was a scholar of the University of London and was awarded a gold medal in physiology. It is to be noted that he also studied physiology at the University of Leipzig under the well-known physiologist Karl Ludwig. In 1889 Martin qualified as a member of the Royal College of Surgeons of England and also became a licentiate of the Society of Apothecaries; the following year he graduated as Bachelor of Medicine in the University of London. Soon after graduation Martin was appointed demonstrator of physiology at the University of Sydney in succession to the late Almroth Wright. After six or seven years he became lecturer in physiology in the University of Melbourne and four years later succeeded to the professorial chair in that subject. During these early stages of his career, Martin studied the formation of antibodies to snake venom. These researches, which were published in the *Journal of the Royal Society of New South Wales* and in the *Journal of Physiology*, brought him before the world of science, and while he was still in Australia in 1901, he was elected a Fellow of the Royal Society. In 1903 he returned to London as Director of the Lister Institute of Preventive Medicine. At this stage we must skip a few years to record Martin's return to Australia in 1931 when he became chief of the Division of Animal Nutrition of the Council for Scientific and Industrial Research in this country. He was also Professor of Biochemistry and General Physiology at the University of Adelaide. Martin had visited Australia in 1923 to attend the Pan-Pacific Science Congress, where he gave a notable address on climate and human efficiency.

The esteem and affection in which Martin was held in Australia were demonstrated clearly when the jubilee of the foundation of the Commonwealth was celebrated early in 1951. The National Health and Medical Research Council addressed to Martin the following memorandum, which was signed by the chairman of the Council, by all its members and by the members of its Research Advisory Committee.

> In this Jubilee year of the Australian Commonwealth, and of your election to the Royal Society, the medical research workers of Australia are honoured to send you their greetings, and to express their admiration and affection.
>
> Your work and teaching in Australian institutions laid a solid foundation to research in this country, and your example and encouragement stimulated its progress during its formative years. Your inspiration still permeates its whole fabric, and you are remembered by Australian workers as one of their most distinguished Masters.
>
> As a tribute to your great work as a scientist and teacher, this Council has established two research fellowships in Medical Science to be known as the Sir Charles James Martin Research Fellowships. These will be awarded periodically to young Australians, to give them overseas experience, and will help to continue the work you encouraged so much.
>
> The undersigned, on behalf of the Australian workers in Medical Science, proffer their warmest remembrances and sincerest greetings.

When the Commonwealth Jubilee Number of THE MEDICAL JOURNAL OF AUSTRALIA was published in January, 1951, a copy was sent to Martin by Sir Percival Hartley, F.R.S. In the issue of April 28, 1951, we published a communication from Sir Percival Hartley in which he quoted part of a letter from Martin. It was noted that 1951 was the Jubilee Year of Sir Charles Martin's Fellowship of the Royal Society. On receiving the Jubilee Number of this journal, Martin wrote as follows.

> I read with pleasure reminiscences of many old colleagues and co-operators in trying to build up a real scientific medical profession in Australia. The material was first rate. The quality of the medical student was superior to what I had been used to teach in London. They all meant business and the experimental method of approach came naturally to them and intellectual adventure was congenial. A drawback for the time being was that the best of them scorned research scholarships after graduation and came to Great Britain or the U.S.A. and were induced to stay. This drainage of some of the best talent was a menace to Australia.

Martin's work at the Lister Institute, which extended over a period of thirty-five years, covered many aspects of medical research. It would be impossible to enumerate these; to do so would necessitate a drawing-up of a complete bibliography. As a director, he was unique. Professor Henry Priestley, in his personal appreciation, has described Martin's attitude to those who worked with him, his constant care for them and the special attention which he always gave to Australians who joined his staff. Not only was Martin director of the Lister Institute; he also, in 1912, became Professor of Experimental Pathology in the University of London. He was also chairman of the committee set up by the War Office to investigate antityphoid inoculation. He took a part in the investigation of plague in India and spent some time in that country in the year 1905.

An interesting chapter might be written on Martin's association with the Australian Imperial Force. Some of the important features only need be mentioned. In the third volume of his "Official History" the late A. Graham Butler refers to the impression made on the analytical medical work of the Australian Imperial Force by the personality "of that great scientist, Sir Charles Martin". Butler describes what happened in the first months of 1916 when the Australian Imperial Force was reconstituted after the Gallipoli campaign. At this stage, A. H. Tebbutt (then major) was appointed "adviser in pathology". Tebbutt drew up a comprehensive proposal for the carrying out of pathological work for the general hospitals and for a mobile laboratory. The story of what Butler calls "the memorable episode" between Tebbutt and Martin has been told before in these pages. Early in March, 1916, the Third Australian General Hospital returned to Egypt from Lemnos, and with it Lieutenant-Colonel C. J. Martin as its pathologist. The situation between Tebbutt and Martin is described by Butler as a delicate one. Lieutenant-Colonel Tebbutt, as he then was, recorded the *dénouement*. Tebbutt went to see Martin and discussed the situation with him. Martin was quite willing to work under Tebbutt, but said that he must draw the line at saluting. With this Tebbutt agreed. These two embarked on the job in hand, but it immediately became apparent to Tebbutt that Martin was the man who should be in charge and Tebbutt asked to be relieved of the post. Martin described what Tebbutt did as "a fine and magnanimous act". Martin undertook investigation of the problems facing the Australian Lighthorsemen and the British Infantry. There had been an outbreak of *cholera asiatica*. Disaster was certain if the disease was allowed to cross the Suez Canal and spread into the crowded population of Egypt. Martin with another officer of the British service formed "diarrhœa camps" and set up a temporary field laboratory in connexion with each camp. The success achieved by these units is described in full by Butler. He also refers to the campaign against malaria in the Jordan Valley and to the "sanitary" improvisations that were also carried out. Later on, Martin, as adviser in pathology, recommended a readjustment of the organization of the pathological services of the Australian Imperial Force to meet the conditions in force in the western theatre in France. He reintroduced the idea of a central research laboratory for the Australian Imperial Force to be stationed in England; he reorganized the pathological service of the general hospitals in France; and he also secured approval for the setting up at No. 3 Australian General Hospital at Abbeville of a laboratory for research designed by himself.

Martin was, of course, a Fellow of the Royal College of Physicians of London. He held the degree of Doctor of Science of the Universities of London and Melbourne. He received the honorary degree of Doctor of Science from Sheffield University and from Trinity College, Dublin. He was an honorary Doctor of Laws of the University of Edinburgh and an honorary Doctor of Civil Law of the University of Durham. Honours were conferred on him. He was twice mentioned in dispatches in connexion with the first World War and received the decoration of C.M.G. He received the Medal of the Royal Society in 1923 and was knighted in 1927. He was appointed a member of the Medical Research Council of Great Britain in 1926. The *British Medical Journal* has recorded the advice and help which he gave to the Association over the years. At the annual meeting in 1908 he was President of the Section of Pathology and two years later he was President of the Section of Bacteriology. He was elected to the Council in 1904 and served more or less continuously until 1916.

We have seen how closely this great man—he was indeed a great man—was associated with Australia and Australians. Many Australians worked with him at the Lister Institute and one and all have difficulty in finding words to describe what he did for them. Australia may well be proud

of the association of Charles Martin with Australian medicine in peace and war, and also thankful for all that he did to aid the progress of medical science.

PROFESSOR W. A. OSBORNE writes: I leave to others better qualified than I am the task of discussing the value of C. J. Martin's research work during the period in which he was Acting Professor of Physiology in the University of Melbourne, also his capacity to inspire pupils and colleagues. I will content myself with one facet of his powers, namely, his highly developed mechanical ability. Martin was in the University of Melbourne in the worst period of its history; the results of the defalcations of a trusted officer combined with a State-wide depression arising from a prolonged drought brought the finances of the university to a low ebb. The utmost economy was exercised; indeed many looking back on that unfortunate time think that economy was overdone and took on the qualities of parsimony. If Martin had inadequate funds allotted to him to buy an instrument he felt was necessary for some investigation, he made it with his own hands. Thus he built up a well-designed and beautifully efficient centrifuge; there was no electricity so he used water power. This centrifuge lasted throughout my time and was extensively used by me. He made from odds and ends his own kymograph and also a pump which allowed him to separate colloids from crystalloids by high-pressure filtration through a clay "candle" impregnated with gelatine. His device for putting a black surface on kymograph paper was the best I have ever seen in any country. His laboratory, sadly cramped in space and equipment, he made efficient for the research work he wished to carry out, and his band of pupils, fortunately small by modern standards, found constant inspiration in his original ideas and his happy faculty of improvising the necessary apparatus. Martin took kindly to Australia, and there can be little doubt that had he been treated more generously he would have remained in the Commonwealth.

O.B. writes: I was privileged to know Sir Charles Martin for more than fifty years; I write far from a reference library but with vivid memories of this great and gifted man.

The remarkable clearness of explanation and the numerous experimental demonstrations are the outstanding memories of the physiology lectures in Melbourne between 1901 and 1903. His wide knowledge of physiology and biochemistry attracted into his laboratory medical seniors such as Stawell, Barrett and Hamilton Russell. Martin carried with him to the Lister Institute in London his affection for Australians, and there one could always count on meeting some keen Australian research worker, Mathieson, Patterson, Burnet, Fairley, Kellaway, Tebbutt or others. But during those years he rendered another great service to our medicine and science, giving unofficial advice when so often consulted on the filling of academic and scientific posts in Australia.

When he was presented with his portrait on retiring from the Lister, Martin said one of his chief aims had been to make the Institute a pleasant and easy place in which to work—and many Australians could testify that work in such company in such well-equipped laboratories was indeed an inspiration, rewarding and pleasant.

Many Australians had reason to be grateful to Martin for his fine work in laboratories and hospitals during the first World War—in Mudros, Egypt and Rouen. Stories were current of his informality. From Mudros he was once called by a "high-up", as the one indispensable expert, to an urgent conference designed to control a grave intestinal outbreak. The General, mellowed and friendly after a mess dinner that night, told Martin he was sure he had met him recently somewhere. After fencing with the remark several times, Martin finally reminded the General that a few days previously he had "keelhauled" him, when, lounging in extremely casual dress on a landing stage, he had failed to salute the General with sufficient snappiness.

Of Martin's sterling work in guiding the destinies of the typhoid and plague inquiries, this is not the place to speak, but one interesting story is not, I believe, in print. After the disheartening results of Wright's anti-typhoid vaccine in the Boer War, the first favourable figures in India so impressed Kitchener that his first reaction was to make vaccination virtually obligatory for all troops in India. This measure would have upset the carefully designed plan, in which Martin was so interested, for gathering invaluable statistically sound figures from regiments where vaccinated volunteers and unvaccinated men were living under identical risks of exposure. It was only by rare tact and influence applied in London through Keogh and others that Martin was able to secure postponement of the order.

His many contributions to our knowledge of nutrition and vitamins were continued to the end of his long working life. When he and his distinguished co-worker, Dame Harriette Chick, were investigating, during the last war, the effects of deprivation of nicotinic acid, I saw Martin at Cambridge himself cleaning out the pens of the experimental pigs in cold, wet and dismal weather. He personally controlled the small wired compound at Cambridge into which he put many wild rabbits and a few infected with myxomatosis—the birth and first application of the idea, so far as I know, of using myxomatosis to combat Australia's greatest pest.

It was characteristic of his approach to a problem that when invited to advise in Australia on animal nutrition and wool production after his retirement from the Lister, he spent some time on his way out, at the excellent laboratories at Onderstepoort in South Africa, to pick up anything useful to Australia.

The Commonwealth, in graceful tribute to his services to Australia, recently established two Sir Charles Martin Research Scholarships and the first holders are, I believe, now at Cambridge.

His long single-minded devotion to science and truth, his inspiring influence as teacher and colleague, his modesty, personal charm and inexhaustible kindness, generosity and ready help will accompany his memory in the hearts of many Australians.

PROFESSOR HENRY PRIESTLEY writes: Anyone who worked with Sir Charles Martin learnt a great deal about many things not directly concerned with the work in hand. I had the great privilege of working with Martin for two years at the Lister Institute. He was a great director of a scientific research institute. He was a master of many branches of medical science and a great administrator. He knew everybody in the Institute intimately and followed every man's work closely and critically. Every few weeks his secretary would say: "Dr. Martin will see you in half an hour." This gave one a little time to collect one's thoughts and facts, for the interview was a very searching one. He would criticize, very severely if necessary, but always, in my experience, pleasantly. I do not think that anyone could have passed through his hands without being better equipped as an investigator. But he did not suffer

fools gladly. He was a most remarkable improviser. I remember asking him for some apparatus. He pointed to a large box in his laboratory and said: "There's the junk box, go and make what you want." Anyone who was on Mudros will remember the wonderful work he did with apparatus made from the most varied refuse.

He was an extraordinarily unselfish person. At a time when the names of heads of departments almost always appeared on every paper published by workers in the departments, Martin's name very seldom appeared, even if he was responsible for the greater part of the mental processes. He was very good to Australian workers who went to London for further experience. When I arrived in London he found a home for me, took me to all sorts of meetings and introduced me to anyone he thought I might like to meet and generally fathered me. Afternoon tea was a feature of the Lister Institute. One was expected to attend these teas when possible. Many important people visited the Institute and whenever he could Martin used to bring them up to tea and then start a discussion. Another way of giving young workers the opportunity to meet and listen to important people was the after dinner meeting. When the Martins had someone of importance to dinner three or four junior workers in the Institute were invited to attend after dinner and listen to and take part in the conversation which was often very stimulating. I had many such happy evenings. Very much of Martin's best work is hidden. For example, he was very largely responsible for the matter of the valuable reports of the Indian Plague Commission which settled the mode of transmission of plague, but his name hardly appears except as a member of the Commission. Much the same applied to many other committees on which he sat.

He was a great man. When I was about to leave for London I met Hinder, then a leading Sydney surgeon, and he said: "So you are going to Charlie Martin. Greatest man in the world. If he thinks you are a ——— fool he will say so."

DR. A. H. TEBBUTT writes: "C.J." covered such a wide field of general and medical science that one felt very humble in his presence, though he obviously liked to be told all of what little one knew on any subject. I count it a great honour to have known him, for no mind of greater distinction have I met. And yet he was a master in the trivial dilemma. We went together to a military depot of medical stores in Egypt. He overheard the somewhat senile officer in charge telling me he had never heard of certain bacteriological items. When he went out into the street C.J. pulled the dulcite and sorbite out of his pocket. He had been nosing around and found the wanted items on a shelf. "He doesn't know he has them and won't miss them." I had occasion to go to the hospital laboratory when he was working and a new technician I had sent him said to me that Major Martin was somewhat queer. He had shown him a rough bottle of bacterial vaccine with many clumps that would not go smooth with shaking by hand. Martin said: "My boy, you don't look well; take the afternoon off. Tie the bottle firmly onto the wheel of a bicycle and have a look at the country round Cairo." His ever inquiring and observant mind led to awkward questions. We drove him up to Palm Beach one afternoon and he picked up a piece of pumice from the flotsam and asked where it came from. To my blank stare he replied that it must have come by sea a long way. I realised then that he was thinking of very distant island volcanoes. Dr. Marjory Little recalls that at the hospital in France where they and Miss Williams worked together he had a cobbler's kit and would repair their shoes in the muddy terrain near Rouen. With all his scientific interests and knowledge he found time to be sympathetic and helpful in a practical way. Those of us who knew him in his prime will ever remember him with affection and admiration.

Correspondence.

REVIEW OF A CANCER DETECTION AND PREVENTION CLINIC.

SIR: May I express my very great interest in the workings of a cancer detection clinic as revealed by Dr. Graham Crawford's review in your journal of April 2, 1955?

At the outset one notices an odd discrepancy in the numerical data. It is stated that over 4000 women have attended, and that at the first visit each woman is interviewed and undergoes a general examination including the taking of a cervical smear for cytological study. In the table of results, the numbers of abnormal and normal smears are set out according to age groups, but the sum of these amounts to 2854.

No less arresting is the number of cases of carcinoma of the cervix (11) claimed to have been found among these seemingly well people. Using the Statistical Register of New South Wales and the life tables published by the Commonwealth Government, one can estimate the yearly death rates due to cancer of the cervix in the age groups of Dr. Crawford's table. Applying these expectations to Dr. Crawford's population of patients, one finds that the expected total deaths due to cancer of cervix in this group is 0·27 persons per year (see table). The death rate due to

Age Group. (Years.)	Estimated Yearly Death Rate Due to Carcinoma of Cervix per 1000 Women.	Patients Attending Clinic.	"Cancers" Discovered in Clinic.	Expected Deaths Due to Genuine Carcinoma of Cervix.
20 to 35	0·013	551	1	0·007
36 to 45	0·050	1208	4	0·060
46 to 55	0·152	748	3	0·113
56 to 65	0·268	286	2	0·077
66 to 80	0·285	61	1	0·017
Total	.. ..		11	0·274

carcinoma of the cervix must be very nearly equal to the incidence rate in similar groups of people. Current treatment could only modify this postulate in so far as it cured carcinoma of the cervix in the sense of complete and permanent eradication of the disease. Such cases of complete and permanent eradication cannot exceed in numbers the ten-year survivors—say 50% of those affected. This leads one to conclude that the yearly incidence of cancer of the cervix in the patients under review is about 0·5 case. Thus it seems that cancer of the cervix has been diagnosed in the cancer detection clinic about 20 times as frequently as the condition can be believed to have genuinely occurred—supposing that, on the average, carcinoma of cervix exists for one year before producing symptoms. (One infers that the cancer detection clinic aims to operate on the basis of annual examination.) This raises more than a strong suspicion that a cancer detection clinic can be a menace to the very safety of the people by "diagnosing" an excessive number of cases of non-existent carcinoma.

There is the further interesting question as to what is attained by the "early" diagnosis of even genuine cancer, but this proposition scarcely calls for review in the present context.

Yours, etc.,

J. R. S. DOUGLAS.
Pathology Department,
The Royal Newcastle Hospital,
Newcastle,
New South Wales.
April 15, 1955.

CLASSIFICATION OF HOSPITAL PATIENTS.

SIR: The effect of the law of New South Wales is to compel medical practitioners in provincial cities and country areas, who have volunteered to treat the needy as honorary medical officers in hospitals, to treat the great majority of the total population as charity patients.

By the schedule for classification of patients issued by the New South Wales Hospitals' Commission (Circular Number 850 of May 22, 1953, with figures corrected by the last basic wage edict), a man earning £1073 16s. a year, who supports a wife and one child and is buying a house at a payment of £5 a week, must be classified as a public patient. So must a bachelor, drawing a salary of £1073 13s. a year and buying a house at £8 10s. per week, be treated as a charity patient.

It would be interesting to see the public's reaction if all carpenters, mechanics, lawyers, accountants and so on were

compelled to give their services free to any employers or clients who demanded them, and who fulfilled the above conditions. I do not imply by this that the medical practitioner should consider his attitude to his work to be on the same plane as these other contributors to the common welfare. He should willingly give his work without payment to the needy.

I say our representatives have failed to tell people that we do so, not only to the needy, but to a majority of the population. I have found that about 85% of fairly wealthy patients so treated are firmly convinced that their doctor received payment from "the Government" for treating them, and so, quite understandably, have no feeling of gratitude for the charity they have received.

Some of these facts were brought to the notice of a member of the New South Wales Branch Council of the British Medical Association by a country local association. His advice to them was that they should try to persuade local hospital lay boards to label more of their beds as "intermediate", so that they would have the privilege enjoyed by the metropolitan honorary of offering the patients immediate hospital treatment in an intermediate or private ward, or the alternative of a waiting time up to many months if they wanted free treatment.

Most members of the local association with whom I have talked disapproved of the long-range effectiveness of this proposal. They feel that a sounder policy would be to try to put our case honestly before the public, and so ultimately to secure a fairer classification schedule of hospital patients. They feel that the method of classification should recognize that many patients conscientiously pay up to £7 10s. a year to a contributory fund, and that they are deprived of much of the benefit of this unless they are comparatively rich and sterile.

A large proportion of country practitioners here is very dissatisfied with the handling of the situation by the New South Wales Branch Council, and feels that a more efficient public relations organization should be obtained.

Yours, etc.,

Lismore, New South Wales, April 30, 1955.

M. R. ROBERTSON.

THE RÖNTGEN ORATION.

SIR: The Röntgen Oration delivered by Mr. J. R. Darling and entitled "On Looking Beneath the Surface of Things" deserves serious consideration. Its main contention that science and religion should be united is being urged by many today—for example, by Mr. Thomas E. Murray, Atomic Energy Commissioner in the United States, in *Time*, April 11 this year, and the Moral Rearmament Brigade, the President of Pakistan, Mr. Nasser of Egypt, the Pope, Mr. Dulles, Sir John Latham *et cetera* in various modes.

The keynote of utterances of this type is: "If the world is too difficult for the scholar to understand, it is by so much the more incomprehensible to the rest of us" (Darling); or "For all you and I know, it may be the incomprehensible and inscrutable will of God to make the twentieth century 'closing time' for the human race" (Thomas).

Therefore it only remains to us to be as like Christ as possible and await our fates. If the earth proves disappointing, tragic and horrible, there is always the next world. This is what science is asked to accept.

Well, there is nothing new or novel about the invitation, but it is certainly daring to extend it at this hour. Since Copernicus's historic testament, science has marched from one victory to another, taking possession of the whole infinite realm of nature, and man today consults physicians, agronomists, meteorologists, astronomers, pathologists, physicists, chemists *et cetera* where a few centuries ago he appealed to God, and scientists have not neglected man and his society, his history, his anatomy, his physiology, his political economy, his ethics.

What is occurring on the earth today from China to Peru presents no incomprehensibility to the scientist, and nothing of which the general line of development was not foreseen. That the main course of these developments is not to the taste of elderly headmasters, clerics, millionaires, politicians *et cetera* and is afflicting to them I do not doubt. Still, realities cannot be dodged or conjured away.

The only course, even if they do not like them, is to adjust themselves to them. If they refuse to recognize this and persist in acting as if things were not so, history gives a deep warning. Like Hitler, Tojo, Mussolini, they will find themselves driven to evil expedients and then to destruction.

The medical profession should be among the first to regret the anti-scientific refurbishing of old nonsense. In their work from day to day physicians, surgeons, oculists *et cetera* use scientific modes of investigation and of treatment. In that sphere they are accustomed to recognize natural law and work accordingly. Surely they should find no difficulty in recognizing that everything on the earth is subject to natural law and that man can only be free to dispose things as he needs them if he understads the natural laws of his society and its evolution, and consequently knows where he is and where he is going.

In medical practice we know that honesty, industry, love of our fellow man, self-sacrifice, decency, temperance are essential to the medical practitioner, but of little avail unless exercised in the full light of scientific investigation and knowledge. These virtues are not supernatural; they are natural and necessary to man as a social individual in this world and in every sphere of his activity only effective if guided by science. Let us reject mysticism, which is merely a cloak for reaction, and stick fast to science.

Yours, etc.,

473 Bourke Street, Melbourne, May 9, 1955.

G. P. O'DAY, M.D.

DOCTORS' NAMES IN THE TELEPHONE DIRECTORY.

SIR: I wish to support Dr. Whitby's remarks on the need for more thought before grouping doctors' names in the "pink pages" under suburbs. The arrangement has been sprung on members without an opportunity for discussion; and as a complete boycott is not likely to be successful without discussion, members will be obliged to comply.

The scheme has evidently been introduced by Council to combat the criticism that doctors cannot be found out of hours. However, the scheme will make no difference to the doctors with solo practices who do not arrange a stand-in, and these men will go off just the same. Those who provide the continuous service will find their burden a little heavier and will have the privilege of paying for it as well. Members who practise near suburb boundaries will doubtless be allowed to pay for a double entry.

I sympathize with Council in their endeavours, but urge that if they introduce the scheme, it should not be at the expense of the profession.

Yours, etc.,

Miranda, New South Wales, May 1, 1955.

J. S. BOXALL.

LUNG CANCER AND SMOKING.

SIR: I am very much indebted to Dr. Orde Poynton for his comments on my letter on this subject (M. J. AUSTRALIA, April 23, 1955). In my letter (M. J. AUSTRALIA, April 2, 1955) I questioned whether neurosis was being needlessly induced in the habitual smoker before the facts were proven to the satisfaction of scientific authority. Dr. Poynton regards the facts as proven. Yet (to quote British authorities alone), the British Empire Cancer Campaign in its thirty-first annual report states that the incrimination of tobacco smoking in lung cancer is not proven. Doll (1953) himself incriminates industrial carcinogens apart from tobacco smoke, and admits that the relatively low mortality from lung cancer in the United States of America is not commensurate with the heavy increase in tobacco consumption in that country.

Not only has Dr. Poynton no doubts about the relationship to lung cancer, but he goes further and states that he is "certain that the risk of cancer of the mouth, nasal sinuses, pharynx, larynx, œsophagus and stomach are all enhanced, and that their incidence would be diminished by abolition of the tobacco habit". Such crusading zeal is admirable, and I am sure that the statement must be based on statistical fact. It is, however, remarkable that Lancaster, in his survey of Australian mortality rates in 1953, showed a drastic fall in buccal cancer from 114 per million in 1910 to 76 per million in 1945. As for laryngeal, œsophageal and

stomach cancer, when the mortality rate in each age group is considered, there is no definite trend in the last thirty-five years in Lancaster's figures. Similarly the Registrar-General's statistical review of England and Wales in 1953 shows no increase in cancer of the lip, tongue, larynx and buccal cavity. Yet, both here and in the United Kingdom, tobacco consumption has increased considerably in the last twenty-five years.

Dr. Poynton does, however, allow that suspicion falls on the exhaust gases of the internal combustion engine. He considers the possible necessity of barring motor traffic from our towns, and replacing them by electrical transport. (Little do the city fathers of Melbourne realize this medical support for their extension of the tram system!) The same carcinogen—benzpyrene—is found in urban road dust, as has been recently identified in the tar of cigarette smoke, but in much higher concentration in the former. I am entirely in agreement with Dr. Poynton that it is the duty of the medical profession to "promote and preserve health regardless of any other consideration and interests that may be involved". I would happily join him in a campaign for cleansing the air of our cities, as well as one for warning the youth of the possible risks of excessive smoking. Either campaign alone should help to reduce the incidence of lung cancer, although recent Press publicity has only stressed the one factor. After all, the smoker takes a risk on his own life alone, whereas the owner of an internal combustion engine and the factory engineer endanger the life of all around, even those innocents who may never have smoked throughout their lives.

Let us preserve a sense of proportion.

Yours, etc.,

Melbourne,
May 2, 1955.

BASIL A. STOLL.

CLASSIFICATION OF RHEUMATOID ARTHRITIS.

SIR: Few will disagree with Dr. Naomi Wing (M. J. AUSTRALIA, April 16) when she says that a patient should be carefully studied before such a label as rheumatoid arthritis is applied. The world-wide new interest in rheumatology has brought forth an international set of standards; men are trying to ensure that the same terms in each country mean the same things.

In our present state of ignorance, however, such terms can only be tentative. Such a word as rheumatoid can be used as we use (for example) carcinoma, to signify a real change in the tissues. Then it is applicable to monarthritis as well as to polyarthritis. The difference between these is as real as the difference between a furuncle and furunculosis.

Or we can use rheumatoid in its popular sense, as a convenient term—like scrofula and dropsy—based on superficial appearances. It is purely tentative, and at the next advance in knowledge may be swept away. But if, as Dr. Wing argues, it should be restricted to a few cases, it becomes meaningless unless it rests on a basis of solid fact by which it can be differentiated from other polyarthritides. At present we do not know if polyarthritis which follows dysentery differs from that which is apparently idiopathic or from any others.

Much of the present confusion in rheumatology has come because our desire to classify has outrun our knowledge. If we are guided by superficial appearances only, we shall be like the two boys who were asked to classify a sack of apples. Not knowing that there existed different kinds of apples (Rome beauties, Jonathans *et cetera*), one graded them into large apples and small apples, and the other into red apples and green apples. Needless to say, both gradings presented a majority of intermediate and unclassifiable types.

Not one of the fifty or so extant classifications of rheumatism can include more than 60% of all cases.[1][2] Like malology,[3] rheumatology presents us with too many intermediate types. For the purposes of communication with our fellows we are condemned to use the terms employed in the classifications. But we should beware lest we attribute to them real meaning; for then we shall be lost in a dream world which has no room for 40% of our patients.

Yours, etc.,

34 Queen's Road,
Melbourne, S.C.2,
May 3, 1955.

M. KELLY.

References.

(1) STONE, K. (1942), "Classification of Rheumatism", *Post Grad. M. J.*, 18: 5.

(2) ROBINSON, W. D., *et alii* (1953), "Rheumatism and Arthritis: Review of American and English Literature of Recent Years (Tenth Rheumatism Review)", *Ann. Int. Med.*, 39: 497.

(3) Malology = the science of apple-grading.

THE CLOSING OF TRUDEAU SANATORIUM.

SIR: On my recent return to Australia from the United States, I have been surprised to hear and to read the inferences in respect to sanatoria in general which have derived from the closing of Trudeau Sanatorium in New York State.

When visiting Trudeau and the research laboratories at Saranac in March of this year, I was privileged to be given by Dr. Mitchell, the Medical Director of Trudeau, full details of all the discussions and considerations which eventually led the Board of the Trudeau Foundation to reach their decision. It may be briefly stated that Trudeau was a unique establishment, built house by house, far from any large centre and in beautiful mountain country, but without any regard to economy in design or efficiency in maintenance or staff economy. For example, each house has its own separate boiler unit, and as there are many separate patients' houses or chalets, the cost of upkeep and the number of staff necessary was fantastic. Inevitably with increasing costs of labour and of foodstuffs, and because of its relative isolation, the costs of maintenance at Trudeau had increased to an astronomical figure.

As Trudeau was a private sanatorium, and in the past few years the State of New York has provided free sanatoria in the State, the demand for private beds had inevitably fallen, and this had not any relationship to any changes in the treatment of tuberculosis, and in fact, it was apparent at Trudeau many years ago that it was such an uneconomical institution that the recent change was inevitable. I was told by Dr. Mitchell that the closure of Trudeau many years ago was postponed only by the use made of it by various contracting authorities such as certain industries.

The purpose of this letter is to suggest that it would be quite erroneous to draw any general conclusions from the fact that Trudeau Sanatorium has been closed, as this was due to local factors only very tenuously related to the treatment of tuberculosis. Certainly it would be quite unfounded to conclude that because of this incident the sanatorium is no longer of value or required in the management of tuberculosis.

Yours, etc.,

85 Spring Street,
Melbourne,
May 7, 1955.

ALAN H. PENINGTON.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

A HOSPITAL FOR GOULBURN.

[From *The Sydney Herald*, January 10, 1833.][1]

To the Editors of *The Sydney Herald*.

Gentlemen,

As an individual concerned in the welfare of Argyle, I am induced to solicit from you the insertion of a few observations on a subject evidently connected with its interests. The inhabitants who, by persevering industry, have raised their country to some consideration in the Colony suffer a few evils which can only be removed by

[1] From the original in the Mitchell Library, Sydney.

the assistance of the Government. The greatest of these is want of a hospital. A country populous as Argyle and the neighbouring counties requires one in some central situation: those who frequent the roads may often observe the agony of invalids travelling to Liverpool. The distance is too far for the conveyance of the sick—it counteracts the purposes of humanity—while it allows full scope for impositions—persons actually requiring medical assistance, in many instances, prefer risking the results at home, rather than obtain relief at such an expense. A servant, wishing to impose upon his master, need only declare himself unwell and give the alternative of being either sent to hospital or allowed to remain idle in his hut. If the former be adopted, he will most probably, by being exposed to the vicissitudes of the weather, during a journey of perhaps 200 miles become so far an invalid, as to elude deserved punishment. It is also known to the experience of every settler, that these characters would have no hesitation in industriously exerting themselves to accomplish such an end. If the latter the employer must ever be dependent on the humour of his servant. Escaping thus, on either hand, with impunity, they are ever tempted to desert him in his greatest emergencies. Being, from this cause, often subject to the absence of their servants, which, even for a day, upon the newly established farms of the interior is a serious loss, I conceive that the settlers should unanimously solicit the Government to erect a hospital on such situation as might be deemed most advantageous. The township of Goulburn appears preferable to any other. This would remedy the evil in a very great degree.

Such a building as would fully answer the purpose might be constructed at a moderate expense, and as it would be conducive to the interests of the settlers I think they ought to contribute. I have to apologize for trespassing thus much upon you—I, of course, merely write as an individual—my remarks may be trifling and inconsiderate. However, they are in some degree warranted by actual experience.

I have, &c.,

M.G.

Argyle,
10 December, 1832.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course at Broken Hill.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that, in conjunction with the Broken Hill Medical Association, a week-end course will be held at the Broken Hill and District Hospital on Saturday and Sunday, June 11 and 12, 1955. The programme is as follows:

Saturday, June 11: 2.30 p.m., "Factors in the Development of Hepatic Cirrhosis", Dr. Bruce Hall; 4 p.m., "The Ovarian Cyst", Dr. Kelvin McGarrity.

Sunday, June 12: 10 a.m., "Cardio-Vascular Surgery", Dr. Harry Windsor; 11.30 a.m., "Cancer of the Alimentary Canal", Dr. Bruce Hall; 2.30 p.m., "Carcinoma of Cervix and Uterus", Dr. Kelvin McGarrity; 4 p.m., "Carcinoma of the Lung", Dr. Harry Windsor.

The fee for attendance at the course will be £5 5s. Those wishing to attend are requested to communicate as soon as possible with Dr. F. Schlink, Honorary Secretary, Broken Hill Medical Association, 252 Mica Street, Broken Hill, telephone number Broken Hill 865.

Examination Results.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the following candidates have satisfied the examiners at the recent examinations for the Diploma in Clinical Pathology: Group I, Monica M. Bullen; Group II, C. W. Kingston, P. K. Lamond; Group III, G. T. Archer, P. K. Lamond, C. J. McDonald.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MAY 7, 1955.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Acute Rheumatism	4	4(4)	..	..	..	..	..	..	8
Amœbiasis	..	..	..	..	4(4)	..	..	..	4
Ancylostomiasis	..	..	10	..	..	..	..	..	10
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	1	..	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	2(1)	14(13)	1(1)	..	1	..	..	..	18
Diphtheria	7(3)	4(4)	3(3)	1(1)	28(28)	1	..	..	44
Dysentery (Bacillary)	..	6(5)	2(2)	..	9(6)	..	..	..	17
Encephalitis	1(1)	..	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	54(19)	37(22)	..	11(9)	4(1)	..	..	..	106
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	1	..	..	..	..	..	1
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	..	1	1(1)	..	..	..	..	..	2
Ophthalmia	..	..	..	..	1	..	..	..	1
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	2	4(3)	9(3)	5(4)	..	..	..	..	20
Puerperal Fever	..	7(5)	..	..	..	..	..	..	7
Rubella	..	..	..	..	1(1)	..	..	..	1
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	18(5)	36(19)	2(1)	13(11)	..	..	..	..	69
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	2	..	1	..	..	..	3
Trachoma	..	..	..	..	13	..	..	..	13
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	18(15)	26(20)	8(2)	9(7)	9(7)	5	..	..	75
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	4(1)	..	..	..	..	..	4
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

[2] Figures not available.

[3] Figures incomplete owing to absence of returns from Northern Territory.

Public Health.

THE POISONS ACT, 1928 (VICTORIA).

THE following additions have been made to the Sixth Schedule to the *Poisons Act*, 1928, by proclamation in the *Victoria Government Gazette*, Number 50, of February 4, 1955.

By His Excellency the Governor of the State of Victoria and its Dependencies in the Commonwealth of Australia, etc.

By virtue of the powers conferred by section 38 of the *Poisons Act*, 1928 (No. 3748), as amended by Act No. 3918, I, the Governor of the State of Victoria in the Commonwealth of Australia, and by and with the advice of the Executive Council of the said State and on the recommendation of the Pharmacy Board of Victoria, do by this my Proclamation amend the Proclamation made by me on the sixteenth day of March, one thousand nine hundred and fifty-five, and published in the *Government Gazette* of the twenty-third day of March, one thousand nine hundred and fifty-five, to amend paragraph (2) of the Sixth Schedule to the said *Poisons Act*, 1928, by the addition thereto of certain substances and preparations such as fluorides of metals, etc., in the manner following, that is to say:

For the expression

> "2. Folic Acid Antagonists such as Teropterin, Aminopterin, Amethopterin, Orthopterin; the solutions, preparations and admixtures of these antagonists or of any of their derivatives by whatever names such folic antagonists are described."

there shall be substituted the expression

> "2. Folic Acid Antagonists such as Teropterin, Aminopterin, Amethopterin, Orthopterin; the solutions, preparations and admixtures of these antagonists or of any of their derivatives by whatever names such folic acid antagonists are described."

Given under my Hand and the Seal of the State of Victoria aforesaid, at Melbourne, this twenty-seventh day of April, in the year of our Lord, one thousand nine hundred and fifty-five, and in the fourth year of the reign of Her Majesty Queen Elizabeth II.

(L.S.) DALLAS BROOKS.

By His Excellency's Command,

V. J. DOUBE,
Minister of Health.

Notice.

VICTORIAN SPORTS MEDICINE ASSOCIATION.

THE inaugural general meeting of the Victorian Sports Medicine Association, which is the Victorian Branch of the Australian Sports Medicine Association, will be held in the Amateur Sports Club of Victoria, McEwan House, 343, Little Collins Street, Melbourne, at 8 p.m. on Wednesday, June 1, 1955. This body will be actively associated with the 1956 Olympic Games, and all members interested in the medical aspects of sport are urged to attend.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Houston, Henry Thomas, M.B., B.S., 1954 (Univ. Sydney), 26 Thorne Street, Ryde, New South Wales.

Reid, James Campbell, M.B., Ch.B. (Glasgow, 1932), F.R.C.S.I. (1951), District Hospital, Broken Hill, New South Wales.

Ewing, Donald Peter, M.B., 1954 (Univ. Sydney), 77 Ocean Street, Woollahra, New South Wales.

The undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Hyde, James O'Halloran, M.B., B.S., 1954 (Univ. Adelaide) (qualified 1953), 7 Azalea Street, Prospect, South Australia.

Lawrence, James Roland, M.B., B.S., 1954 (Univ. Adelaide) (qualified 1953), 1 View Street, Walkerville, South Australia.

Hoile, Edward Murray, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952), Kingscote, Kangaroo Island, South Australia.

Deaths.

THE following deaths have been announced:

HENDERSON.—James Mann Henderson, on May 9, 1955, at Melbourne.

BYRNE.—James Michael Byrne, on May 17, 1955, at Ramsgate, New South Wales.

Diary for the Month.

JUNE 1.—Western Australian Branch, B.M.A.: Branch Council.
JUNE 3.—Queensland Branch, B.M.A.: General Meeting.
JUNE 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 10.—Tasmanian Branch, B.M.A.: Branch Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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